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The Jackson Lecture.[1]

MEDICAL HISTORY—DO WE USE IT WISELY?

By R. A. O'BRIEN, C.B.E., M.D.,

Queensland Institute of Medical Research, Brisbane.

IT is first my pleasant duty to thank the Queensland Branch of the British Medical Association for the honour of its invitation to join the illustrious list of Jackson lecturers.

Presumably it is not usual to commence an occasion such as this with prayer; but perhaps it is not inappropriate tonight to recall the noble Bidding Prayer based on the Apocrypha, intoned daily or weekly in so many schools and colleges: "Let us now praise famous men . . . particularly the benefactors of this school . . . and let us pray for all seminaries of sound learning and especially the universities of this land." And there were famous men in Brisbane in the days I recall. I was privileged to come in contact with Jackson, John Thompson, Wilton Love, Jefferis Turner and James O'Neil Mayne; from one and all we neophytes received ceaseless kindness and help, and for that I felt, and continue to feel, deeply grateful. To check my memory of their work, I sought the old records of the Brisbane Hospital immediately after 1902, when I was at the hospital. Failing to find these records in any public or private library in Brisbane, I visited the Mitchell Library in Sydney, where the librarian with prompt courtesy immediately produced a series of the old reports. There stands evidence, viewed in the light of medicine and surgery I have since seen in many centres in the old world, that the surgery of Jackson and Thompson, the beautiful bacteriological photography of John Thompson, the medicine of Jefferis Turner and Wilton Love, and the wide pathological knowledge and competence of the latter, gave Brisbane a team of remarkably high standard.

I am imagining tonight that I was privileged to share with Jackson the imaginary Rip Van Winkle experience of knowing Brisbane and the Brisbane General Hospital in the period 1902 to 1905 and not seeing it again until a revisit in 1949 to survey the field.

Jackson cast his eye first to the slope behind Ward 9, to seek for Dr. Mayne's loved small garden of beauty containing perhaps one hundred kinds of wattle trees, and, not finding any of the trees, he turned with relief to recognize an old friend apparently unchanged—the authentic ancient morgue. We debated—with gruesome memories of watching blood-wet fingers turning the only tap available for handwashing in the morgue—how any of us escaped infection. I recall assisting at autopsies of patients who had died in less than twenty-four hours after the first symptoms of virulent septicæmic or pneumonic plague. We had no rubber gloves in those days but trusted to soap. We did not all escape. The hospital report for 1902 recalls that Dr. Maclean, acting superintendent, suffered a severe attack of bubonic plague contracted in the hospital. In 1900, the morgue attendant James Jackson, died of bubonic plague, and in 1902 Dr. C. J. Hall Wray, who performed many autopsies on plague patients, himself "contracted the disease in the post-mortem room" and died (Halford, 1902).

After an astonished gaze at the prodigious pile of buildings that had arisen, Jackson made his way first to the operating theatre unit, noting, amongst many new features, autoclaves, for there were no autoclaves in the hospital in 1903.

[1] Delivered at a meeting of the Queensland Branch of the British Medical Association on September 2, 1949.

After answering Jackson's questions I explained that my recent interest was due to an invitation by the Department of Health and Home Affairs to investigate autoclaves in Brisbane hospitals; this arose from deaths due to tetanus and gas gangrene following "clean" operations. As the result of several months' work, I found the paradox of high efficiency at some points with depressingly poor performance at others.

We discussed the conditions needed for sterilization and for efficient operation of autoclaves (Spooner and Turnbull, 1942; "Annotation", 1939; Underwood, 1934; Walter, 1948). For sterilization of ordinary (non-fatty) dressings, instruments, *et cetera*, with saturated steam, the requirements are clear. (i) A temperature of 120° C. must be reached and maintained in every corner and recess of dressings, utensils, instruments *et cetera*, for a safe period, defined by most workers as fifteen to twenty minutes at a pressure of 20 pounds. (ii) To attain 120° C. at this pressure, all the air or approximately all must be removed from the sterilizing chamber by (*a*) swirling steam, or (*b*) repeated production of a "vacuum" of at least 10 inches of mercury, or (*c*) a "discharge line" at the bottom of the autoclave with an adjustable thermostatic valve. (iii) The saturated steam must be allowed to circulate freely and to penetrate all materials. In

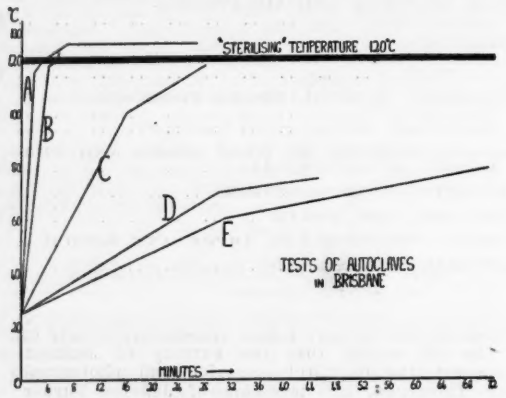

FIGURE I.
Autoclave results.

brief, the bundles of dressings must be small, they must be packed very loosely on their sides, utensils must be so placed as to exclude pockets of still air, preferably on their sides. The packages of dressings and instruments must allow free circulation of steam—about one-third of the space should be unoccupied.

The curves in Figure I illustrate some of the results. Hospital B had steam laid on; the jacket was always kept warm with steam at 20 pounds; the "vacuum" of twelve inches was reached in three minutes; steam at 20 pounds pressure was admitted at the top of the chamber; the discharge line was at the bottom; there was an indicating thermometer at the exit from the chamber and the thermostatic valve was satisfactory; the bundles were small, placed in the chamber on their sides; approximately one-third of the total space of the chamber was unoccupied, so that ample circulation of steam was permitted. The results were highly satisfactory: the desired temperature of 120° C. was reached within four minutes of closing of the door of the chamber, and was maintained. By use of the "vacuum" and by slightly opening the door of the chamber at the end of sterilization for five to ten minutes good drying was obtained.

Hospitals C, D and E provided illustrations of breaches of most of the fundamental rules—and their results. In C the autoclaves were provided with a poor heating system, for the temperature took about thirty minutes to reach 120° C. in the chamber; the temperature taken inside a roll of dressings in the drum normally in use in this hospital gave a reading of 5° below this figure.

In Hospital D we found deficient heating and heavy overcrowding in the chamber, with huge bundles of dressings. After forty-five minutes only 75° C. had been reached.

In Hospital E deficient heating was present: after seventy minutes the temperature was below 90° C.

After these disconcerting results had been recorded, it was pleasant to know that a promising autoclave was then designed and made by a Brisbane firm. At a later date we tested this installation, which proved to be strikingly efficient. Curve A (Figure I) records that the thermocouple inside the chamber registered 120° C. within four minutes of turning on of the electric current in the heaters. (Is one allowed to wonder whether a similar investigation—with the self-recording mercury thermometers, the benzoic-acid-methylene-blue tubes and the thermocouple, arranged and used by my colleague, Mr Callaghan—in the hospitals in the other capitals of Australia would yield results even distantly resembling those noted by Mr. Callaghan and myself in Brisbane?)

Here we may pause a moment to consider the query which will haunt us throughout: do we learn enough, and rapidly enough, from medical history? Is there an avoidable lag in the application of available knowledge? The following three quotations may make us ponder.

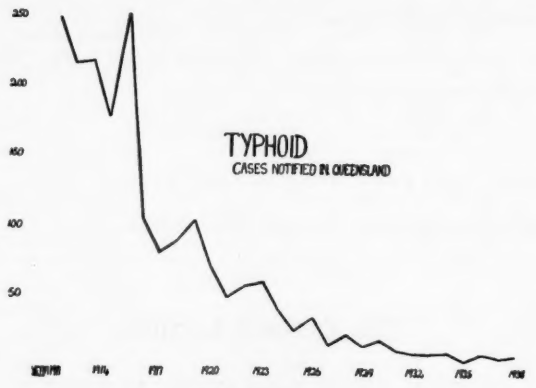

FIGURE II.
Typhoid fever cases in Queensland.

The first is from Walter (1948):

> The detrimental effect of air in the sterilizer (Rubner, 1898), the hazard of superheating (Esmarck, 1888) . . . the advantage of gravity air clearance, the influence of sterilizer design on efficiency, the necessity of measuring the bactericidal power of "steam" by taking its temperature (Frosch, 1890)—all were recognised long ago. Schimmelbusch in 1891 advocated boiling nail-brushes in 1% Na_2CO_3.

The second is also from Walter (1948):

> A steam "sterilizer" of the Arnold pattern was originally designed for the cooking of vegetables without immersion in water. It was then adopted for "sterilization" of hospital supplies and it is still used in remote places.

Observe the three last words. Such steamers, called by an irreverent colleague "pie-warmers", were in widespread use here as "sterilizers" prior to action by the Department of Health and Home Affairs about a year ago.

The third is an "Annotation" (1942):

> We have referred on several recent occasions to warnings by various authors that sterilization by steam under pressure has by no means the cast-iron reliability usually credited to it. Failure may be due either to misuse, which means inadequate air discharge, or unduly tight and massive packing of the material to be sterilized, or to faulty design of the autoclave itself. The discovery that steam sterilizing apparatus is not functioning properly is made in various places from time to time, usually causing consternation.

Yet, in 1948, this same consternation was caused in Brisbane by a rediscovery of the same discovery.

The next feature to strike Jackson's attention was the transformation of the activities of Wards 9 and 10. When Jackson was superintendent, and later, when I was a resident medical officer, we expected these wards each summer to house 100 to 250 patients suffering from typhoid fever. Now they are empty of typhoid patients. Three thoughts quickly presented themselves: (i) the astonishing reduction in the number of typhoid patients treated; (ii) in retrospect, the remarkably low incidence of typhoid fever amongst the nurses; and (iii) the low incidence among the doctors attending these patients.

Figure II shows the number of cases reported in Queensland from 1911 to 1938.

It is not often that the hygienist, immersed in the daily repellent task of unromantic inspection of garbage heaps, odorous sewers *et cetera*, has the deep spiritual satisfaction of watching his efforts produce dramatic results; but I think the reduction of typhoid fever in Queensland (without travelling further afield) is a conspicuous instance of the beneficial result that can follow sustained enthusiasm and effort directed by intelligence and knowledge regardless of the jibes of those with little vision. And here let me say that I noted with deep satisfaction, on the wall of the present Chief Sanitary Inspector's office, the photograph of my old friend John Simpson, the Chief Sanitary Inspector of that early period, to whom the Department of Health of Queensland owed so much—pleasant proof that succeeding generations are not always unmindful of worthy men in the past.

In surveying the great achievement of the Department of Health, may I briefly glance at history? In 1903, Dr. B. B. Ham, the former medical head of the health department, wrote: "The loathsome cesspit with its contents putrefying for months and even for years near dwelling houses." In 1902-1904 1400 cases of typhoid fever were attributed to "drought with resulting contaminations of sparse water supplies, fly carriage of infected material, infected dust blown off roofs and then into tanks, and pollution of well water from the cesspits". In 1904 the advice was given to "keep closet seats covered", "use disinfectant to discourage flies from breeding", "build outhouses properly to exclude rats and flies". But we can never rest content with our achievement. The alert health department can never forget that the typhoid bacillus often has a long innings and that even now there must exist "carriers" in this State. Chicago reports for 1948 (*The Journal of the American Medical Association*) record that a woman carrier who had been under observation for fourteen years concluded that, since tests had given negative results, she was no longer a carrier. She therefore arranged a family dinner. Her own children had been immunized with typhoid vaccine earlier when it was first discovered that she was a carrier, and they had been reimmunized later. The unprotected son-in-law and daughter-in-law attended the dinner. Both developed typhoid fever within two weeks. Her daughter-in-law infected a sister, who in her turn infected her own three children. The story provides startling confirmation of the hygienist's traditional distrust of the personal habits of cooks and of the bacterial population of their hands. In Gympie (Director-General of Health and Medical Services, 1937-1938) a few years ago, a carrier, twelve years after her attack of typhoid fever, infected six people.

I mentioned the low incidence of typhoid fever amongst nurses and doctors in the Brisbane General Hospital in the early days. This rarity of attack to me is inexplicable, for the nurses in the male and female typhoid wards had unlimited chances of infection. Brisbane had no water-carriage system of sewerage; the pan system was in use at the hospital and flies were never absent. Anyone who has seen the urine of a typhoid patient shimmering with countless millions of typhoid bacilli must wonder that any nurses in those days could escape infection. We wore no rubber gloves; we had no apparatus for heat sterilization of the bed pans or urine bottles. Carbolic acid was available, but my memory is that the utensils soiled with fæces and urine were only occasionally cleaned with an antiseptic. I remember well that one honorary physician created consternation by requiring a time and quantity chart of all urine passed by all febrile typhoid patients. And how the bath men escaped infection puzzles me. In the "bath treatment" (Figure III), highly valued by nurses and doctors alike, each patient with pyrexia was immersed in a huge portable bath several times a day, even three-hourly. I am fairly certain that no antiseptic was used in the water, but I am quite certain that any antiseptic that may have been present was not sufficient to kill typhoid bacilli on the patient's perineum in the short exposure during the bath; and yet in the three or four years of my acquaintance with these wards I know of no infection of a bath attendant. I cannot find in the records available any note of the number of nurses infected. My memory is that each summer typhoid season one or perhaps two nurses became infected, but none of

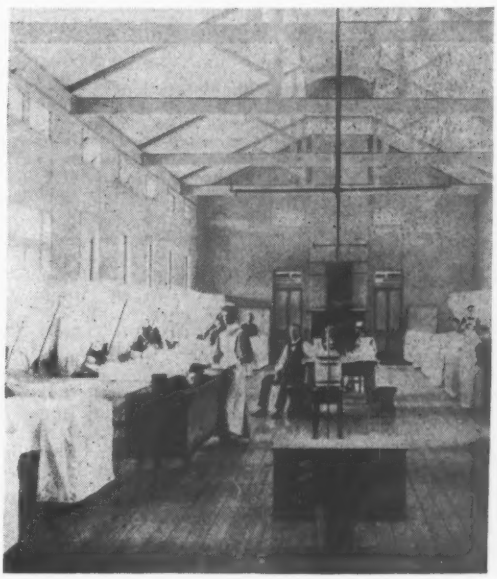

FIGURE III.
A portable bath.

the doctors. In the decade of which some details are still available only one nurse died of typhoid fever, and that attack was attributed to ward infection.

A supposition that presents itself is that we all suffered sub-clinical attacks of typhoid—possibly a trifling transient diarrhœa—and then developed sufficient immunity to protect us during the remainder of our exposure. Unfortunately this hypothesis cannot now be tested.

In contrast to that period, today a single admission to hospital of a typhoid patient causes comment throughout the hospital, and nurses and doctors are well guarded against risk of infection.

Yet here again it is salutary to contemplate for a moment the curious manifestations of delay, procrastination, time-lag in matters of medical history. The brilliant success of the campaign against typhoid fever, patiently carried on here over a number of years, is marred only by the reflection that the essential knowledge of the danger of fly carriage had been common property since the Americans paid for the acquirement of that knowledge by 20,000 cases of typhoid fever amongst 107,000 troops in camp in the southern United States of America during the war with Spain in 1898 (Rosenau, 1913).

I was asked to go to Charters Towers about 1903 to examine a typhoid outbreak there, and I well remember

the discussions on my report. They ranged round many of the requirements we recognize today as vital—for example, comprehensive anti-fly measures, the abolition of cesspits, the proper rat-proof and fly-proof construction of earth closets, the use of proper metal pans where sewerage does not exist, and proper disposal of horse-dung. (Vaccination was unknown.)

On this disconcerting point of lag, Dr. J. H. L. Cumpston, the Australian Director-General of Health, wrote (1927): "Although the relation between typhoid fever and absence of ordered sanitation was soon perceived, it was not properly accepted as the basis for official action until about fifty years after the knowledge was available."

We may further contrast the immediate acceptance of penicillin and insulin by the medical world and the clamour for them by the lay public, with the general neglect and procrastination where preventive medicine —insurance for the distant future—is concerned. Two instances that spring to my mind at once relate to the risk from milk laden with tubercle bacilli and to immunization against diphtheria. The great German chemist, Soxhlet, in 1880 recommended the heating or boiling of milk destined for infant food, and it is stated that the practice was widely adopted in Austria and Germany with resultant reduction in infant mortality. I tested London milk for the London County Council in 1910, and found that about 13% of churns reaching London railway stations contained pathogenic tubercle bacilli. Yet, in 1942, thirty-two years later, the Committee on Tuberculosis in War-Time reported that 40% of the cattle in Britain reacted to tuberculin, and a few years ago pathogenic tubercule bacilli could be found in almost every 3000-gallon railway milk tank coming into the larger cities. G. S. Wilson (1949) estimated that milk infected with pathogenic tubercle caused 1500 deaths each year in England.

The most outstanding example of lag in my own experience is connected with immunization against diphtheria. Before the 1914-1918 war, the idea of immunization of children against diphtheria had just made its appearance in German medical literature. During the war, Park and Zingher in New York tested the method and established its usefulness. I went to New York in 1920 and saw the results of their successful work. In 1921, after long delay, we succeeded in obtaining permission to try immunization at a residential school of boys and girls in London. In 1922 we published the first paper in England showing that by development of the American methods and our own improvements it was possible safely to immunize children and to give them almost complete protection against diphtheria. At that time the disease was still causing 60,000 cases a year in England and 3000 deaths. Though Harries and other heads of infectious fevers hospitals soon, by active immunization, almost abolished diphtheria from their staffs, and although a few progressive local health authorities produced a local reduction in the incidence of diphtheria, the numbers of cases and deaths—55,000 and 2800—remained almost stationary until early in the recent war, when Sir Wilson Jamieson, who had been taken into the Ministry of Health, quickly planned a comprehensive scheme of active immunization. This campaign was financed by the Ministry and soon led to the striking reduction in incidence and mortality confidently expected. In 1948 the reported cases numbered 8034, deaths 150. Here the lag was approximately twenty years.

The story of the pressure cooker provides another amusing instance of lag. At the end of the seventeenth century Denys Papin invented a number of ingenious mechanical contrivances, amongst them a steam pressure "New Digester" in 1681 (Figure IV). It was heralded, we are told, with grandiloquent promises of an immediate gastronomic Elysium, however tough the viands. Yet it is only within the past decade that the pressure cooker, that boon for the busy goddess of the kitchen, has become widely known and used in America, Britain and Australia. (In passing, one can readily show, with capillaries of benzoic acid powder which melts at 120° C., that the pressure cooker can be so used as to sterilize syringes and small emergency instruments.)

In this voyage with Jackson, we discussed another activity dear to his heart, the medical education of nurses and students. As we know, Jackson worked—and successfully—for years to establish and maintain a teaching school for Queensland nurses. It is not our business tonight to debate the medical curriculum. I am concerned with the wider problem of what may be called, for want of clearer terms, "historical background" or "culture". The golden thread which runs through the Jackson lectures is that of history. A medical student recently remarked to me on the difference between the life of a medical student in England or Europe and one in Australia. In the northern hemisphere the sense of history is ever present. In their daily journey students pass halls,

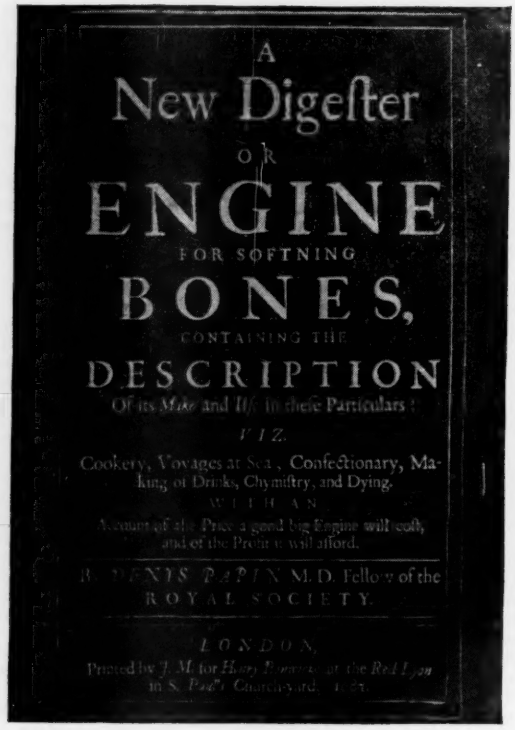
A
New Digeſter
OR
ENGINE
FOR SOFTNING
BONES,
CONTAINING THE
DESCRIPTION
Of its Make and Uſe in theſe Particulars:
VIZ.
Cookery, Voyages at Sea, Confectionary, Making of Drinks, Chymiſtry, and Dying.
WITH AN
Account of the Price a good big Engine will coſt, and of the Profit it will afford.
DENYS PAPIN M.D. Fellow of the ROYAL SOCIETY.
LONDON,
Printed by J. M. for Henry Bonwick at the Red Lyon in S. Paul's Churchyard. 1681.

FIGURE IV.

Denys Papin, "A New Digester"; photograph by courtesy of the Wellcome Historical Medical Museum.

theatres, churches or cathedrals whose roots run back through the centuries. Here in this new land at first sight we have nothing to compare. But, though the student in Queensland cannot turn these pregnant pages of time by gazing at the Sphinx, walking in Pompeii, or thrilling over the light and shade of a Norman doorway, there is still a world of historic enjoyment here.

The primeval marsupial takes us far enough back. Today I examined the 13 smooth-trunked gum trees in front of this medical school, and on every tree I found the marks of recent visits by opossums. On one tree I found scores of marks—evidence of high-jinks on moonlight nights in the very University precincts. And if we tire of modernity, I would suggest a bus-ride to Lone Pine; when we see there a tree kangaroo climb 40 feet up a eucalypt, or hear how the newborn kangaroo or wallaby finds its immature way to the pouch and teat, which immediately swells and holds the young thing fixed and safe at the source of food for weeks, we may rightly feel for a moment that the London student passing the site of an early Saxon victory, a Roman bath or a statue of Boadicea, has not everything his own way. Nor can the

London student offer to show a visitor four score pelicans a few miles from his medical school—as we can on the sandbank at Lytton, or, as a professional fisherman recently assured me, 200 black swans feeding on sea-grass near the Pile Light. And even here, if we keep our ears attuned, we may hear magic murmurs of the realms of gold. During the centenary celebrations of Fortitude Valley recently, we learned that one of the first few ships to bring the earliest Valley settlers was *Artemisia*. I remember that Artemisia was the widow of King Mausoleus, in whose memory she built one of the seven wonders of the ancient world—"The Mausoleum". I then sought out her record in the encyclopædia. A few evenings later, during a "walkie-talkie" broadcast from the aboriginal settlement of Delissaville in the Northern Territory, the narrator told, as we heard the sorrowful wailing, how the chief mourner at the ceremony, in memory of a recently dead aboriginal woman, burnt all the belongings of the departed in a ceremonial fire. He then dug a small hole and buried almost all the available ashes, again with incantations. Then with wonderment I heard the narrator describe how the chief actor made a small damper, sprinkled it with the remaining ashes of the departed, cooked it on the hot stone in the sacred area and ate it. It gave me a momentary thrill to recall that Queen Artemisia showed the same regard for her dead lord by putting his ashes in a glass of wine and solemnly drinking the potion.

It amused me to recall to Jackson's memory a sunny hour, almost on the ground on which this building now stands. In 1902 we had as a patient in the hospital a full-blooded aboriginal called Charlie. He had chronic groin ulceration needing periodic cauterization. Between whiles, Charlie functioned as an unofficial member of the hospital staff, carrying round the baskets of medicine and stores from ward to ward. He was a favourite with everyone and knew every happening in the hospital. Did Jackson or Mayne scold an "R.M.O.", Charlie knew; was Matron's temper frayed that day, Charlie knew; did an "R.M.O." and an attractive nurse meet outside the hospital, Charlie knew.

Against that background consider this scene. Dr. Walter Roth wished to replace a broken photographic plate showing the *Mangani* ceremony of pointing the bone (Figure V). Roth, Charlie and I climbed over the back fence of the hospital and walked near the top of the hill to a suitable spot just about where this medical school stands. Charlie stripped and posed for the photograph which Roth took. I made some casual remark to Roth of wonder that the medicine man, master of arts and craft, had been able to succeed in his pretended abstraction of the distant victim's blood and was never detected, whereupon the sophisticated Charlie took me seriously to task, exclaiming with earnest face and deep emphasis: "My word, doctor, that plenty fellow true, *Mangani* kill him every time." (Amongst the fine bronze reliefs of aboriginal life on the doors of the Public Library of New South Wales in Sydney is an excellent representation of "pointing the bone"—there called *Irma*—the string of human hair being carefully held clear of the ground by the assistant during the ceremony.) In the doings of this primitive folk we have our own earliest dawnings of fetishism, totemism and religion, and the engrossing story of Fraser's "Golden Bough".

And if we smile in our 1949 wisdom at such aboriginal simplicities, it may be salutary for us to remember that "two hundred years ago practically everybody believed in spooks and demons. The last legal execution for witchcraft occurred late in the nineteenth century so that our grandfathers could have known people who had seen men and women put to death for associating with the Devil". Blackstone, the great legal commentator, said that "to deny the actual existence of witchcraft is flatly to contradict the revealed word of God". Wesley to the end of his life maintained that "the giving up of a belief in witchcraft is in effect giving up the Bible".

When I took over the Plague Isolation Hospital at Colmslie in 1904, there were in the administrative room two telephones at the extreme ends of the room—one to the wards and one to Brisbane. When inquiries came through from Brisbane we had to cross the room to inquire from the wards. I asked why this curiously inefficient arrangement was made. I was assurred by the nurse (and the tale was later repeated by an official of our Department of Health) that it was to quieten the minds of the citizens of Brisbane and assure them that every precaution had been taken to protect them against any danger of transmitting infection. I am still sure that, whether the explanation was true or no, those two folk, in a position to know, believed it.

While discussing the numbers of patients available to medical students in the hospital, Jackson and I happened on an arresting extract in an old Brisbane Hospital report—oddly enough, a quotation from "Utopia" (1516):

> In the city they have hospitals so ample, which were devised to the intent that the sick, be they never so many, should not be too throng or straight . . . and partly that they that were taken with diseases which be wont to creep from one to another might be laid far apart.

I felt then that if I were the harassed administrative head of an overcrowded hospital with many of its beds within 40, 30, and even 20 inches of each other, I would

FIGURE V.
The *Mangani* ceremony.

feel impelled to ask myself whether my task had been made more arduous because perchance we had not learnt enough from medical history and not learnt quickly enough. It is forty-seven years since that note was published in the Brisbane Hospital report.

I reminded Jackson, in our hypothetical journey, of one occasion in 1902, when he pointed out to me the contrast between a ward patient suffering from pernicious anæmia and one with chlorosis. This naturally led our talk to the present position of blood diseases in Queensland.

It has been my pleasant privilege during the past year, thanks to the courtesy of Dr. A. D. D. Pye, Dr. J. V. Duhig and Dr. G. C. Taylor, to consider "blood diseases" in Queensland, and particularly at the Brisbane General Hospital. One seeming difference between local and English conditions struck me. We had recently arranged a simple halometer with two electric bulbs, and were surprised to find that more than 1000 slides from adults and adolescents sent for blood examination did not yield one indisputable finding of microcytic anæmia. Apparently chronic microcytic anæmia due to iron starvation is rare in Brisbane. Impressed by this—for these anæmias were met with not uncommonly in London—I asked about koilonychia or Plummer-Vinson (Paterson's) syndrome. Dr. Duhig told me that in thirty years in Brisbane he had seen only one patient with koilonychia (she also showed Plummer-Vinson syndrome). Amongst about 800 midwifery

patients at the Brisbane Women's Hospital examined since the laboratory opened, in not more than three or four cases has microcytosis been observable in stained blood films. In greatest contrast with this is Stanley Davidson's report (1933) that about 50 in every 100 women of the poorest classes in Aberdeen suffered from chronic iron-deficiency anæmia, due to losses at menstruation and parturition, to frequent pregnancies and to inadequate diet. I am not clear about the occurrence of this disease in Sydney and Melbourne. Dr. A. H. Tebbutt kindly looked through some of his records and found a few instances of low mean cell hæmoglobin concentration. At two of the large Sydney hospitals, apparently, true chronic microcytic iron-deficiency anæmia is rare; but Dr. E. S. J. King, of the Royal Melbourne Hospital, kindly told me that he sees a few patients with Plummer-Vinson syndrome every year and about twice a month an undoubted instance of iron-deficiency anæmia.

Dr. Humble, of the Westminster Hospital in London, told me recently that amongst some 5000 patients examined in the hæmatological department each year he finds about eight or ten with microcytic iron-deficiency anæmia.

Dr. Mollin, from the Postgraduate School in London, wrote that from four to 12 cases of this disease and one or two of koilonychia occur each year in the practice of the hospital of 500 beds.

Probably the percentage of people suffering from inadequate diet in Australia is lower than anywhere in the world, and the comparative rarity of this anæmia can be simply explained by the luxurious Australian diet and high consumption of meat; but the point merits further inquiry.

My original aim was to inquire whether the common "diseases of the blood"—the leuchæmias, the purpuras and pernicious anæmia—were increasing or not in Queensland. After spending many days over the filed records made available by the kindness of Dr. Pye, I regretfully felt compelled to give up the unpromising quest. It soon became clear that the data were insufficient to enable one to assign the case-sheets to satisfactory groups—acute or chronic, myeloid or lymphatic leuchæmia, *et cetera* (or even leuchæmia of any kind).

The graph in Figure VI gives the crude figures for deaths from "leuchæmia" in Queensland, which the Registrar-General kindly gave me; the rate of increase seems alarming, but I do not know what importance is to be attached to these uncorrected figures. Hæmatologists in England and America believe that leuchæmia is increasing, and Dr. E. S. J. King, of the Royal Melbourne Hospital, told me that careful checking of slides and records has convinced him that the number of patients each year suffering from leuchæmia in Victoria has increased.

My experience with pernicious anæmia was equally disconcerting. After spending many hours trying to divide the case reports into groups—those with enough data to satisfy rigid diagnostic criteria, the "probables", the "possibles", and the "clearly misdiagnosed"—I abandoned the attempt because of insufficient data in the case-sheets. Dr. R. J. Sawers, registrar to the professor of medicine, has been braver; he has recently analysed the records of all patients who received injections of liver extract in one month (approximately) at the Brisbane General Hospital. His findings were, in general, so like my own that I asked him if I might use them as presenting a fair picture. During the month 242 patients received injections of liver extract in the out-patient department of the Brisbane General Hospital. Since no case was labelled "macrocytic nutritional anæmia" or "macrocytic anæmia of pregnancy", we shall not introduce any great error into the discussion if we conclude that all the 242 patients were being given injections of liver extract because of the belief that they were suffering from pernicious anæmia.

Of the 242, "the records of 38 were not found on first search"—an unpromising beginning; 67 were "not adequately documented to allow conclusions"; "44 showed in their records no response to treatment or a response not attributable to liver injections". There remained 97.

On what basis shall we grade these? A consulting physician in a modern teaching hospital, before presenting a patient to his medical class as a fully-documented case of pernicious anæmia, would discuss the following evidence: megaloblastic marrow, macrocytic hyperchromic anæmia, histamine-resistant achlorhydria, the clinical syndrome of pernicious anæmia (colour, tongue, *et cetera*), the presence or absence of signs or symptoms of subacute combined degeneration of the cord, particularly any diminution of vibration sense. He would, if there was no evidence of neurological disease, inject one dose of potent liver extract —for example, one millilitre of "Examen" or "Reticulogen" or two millilitres of "Anahæmin"—and would expect approximately the reticulocyte rise demanded by Riddle's equation (Dyke, 1947); and on giving one further dose after a fortnight, he would expect to find two weeks later in the average patient a rise in red cells satisfying the Della Vida formula. (He would, incidentally, thus get a valuable indication of the potency of the liver extract he was using.)

It is a reasonable assumption that the consultant or professor of medicine presented with the type of evidence which Dr. Sawers and I found in the records here would accept few of the 97—possibly three—as satisfying all criteria. If the consultant does not demand for diagnosis the marrow picture, he still must hesitate over a considerable number of the 94, for Dr. Sawers records that no

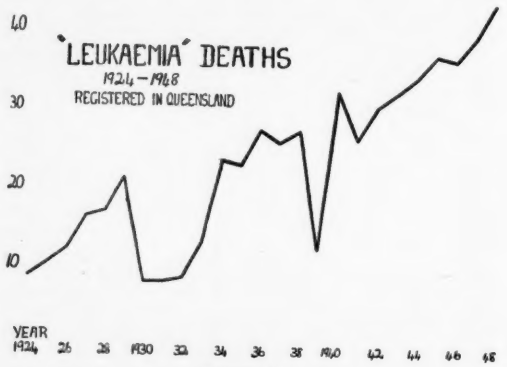

FIGURE VI.

Deaths from leuchæmia registered in Queensland.

test for achlorhydria was made in over 50% of the 97 cases, no test was made for a reticulocyte rise or increase of hæmoglobin value in "nearly 50%", and in several instances no blood examination had been made for as long as five years.

At all events, after classifying the case records under various headings, Dr. Sawers evolved a series of marks, four representing almost full diagnostic evidence. In this class fell three patients of the 97, 38 got three marks, 38 got two, and 16 got one.

The question here suggests itself: Why should we at the Brisbane General Hospital accept a lowered standard of diagnosis? What of the case records that received two marks or one? We all would wish our standards reached in the daily round at the Brisbane General Hospital to be equal to those of the ordinary London teaching hospital or those of Sydney or Melbourne schools—I am told that there are some who believe they are. Just before I left London I attended a meeting addressed by one of the foremost English hæmatologists. During question-time after the lecture I said that Dameshek had estimated the "wastage" of liver extract, because of unnecessary administration or unduly high dosage, as being between one-half and two-thirds of that used in the United States of America. I asked the lecturer's opinion of wastage in England. He said that, answering on the spur of the moment and without figures, he would put the wastage of liver extract in general practice even higher than the American estimate; in the teaching hospitals the wastage would be negligible. One is driven to speculate on what

figure an adequate hæmatological department would find for "wastage" in the several thousand pounds a year that liver extract must cost the Brisbane General Hospital.

Concerning the 44 patients who did not respond to treatment or whose improvement was not attributable to liver treatment, I doubt if a committee of the senior physicians of the Brisbane General Hospital would unhesitatingly accept one as a sufferer from pernicious anæmia. I have persuaded Dr. Sawers to allow me to quote extracts from his general analysis and comment: "Liver extract is given frequently without investigation." "No record of tests with tuning-fork was found except at one O.P. clinic." "Diagnosis of the causes of anæmia in the hospital could be improved." Perhaps this final meiosis may be accepted as a strand in the plea for a hæmatological department and staff comparable with those in the large southern or London hospitals.

As Dr. Duhig has repeatedly pointed out, his equipment and staff—who struggle in a most praiseworthy fashion with discouraging pressure of work—cannot deal with all the blood examinations as satisfactorily as he would wish. In an attempt to estimate what would be a reasonable complement for the hæmatological department of the Brisbane General Hospital, some difficulty arises, for it is fairly certain that huge teaching hospitals of 1200 to 1400 bed size are things of the past in England. With the exception of the Edinburgh Royal Infirmary, very few of the best-known teaching hospitals in England have more than 500 to 700 beds. Several English hæmatologists who have been good enough to give me their opinions estimate that a hospital of the size of the Brisbane General Hospital would require for the department of hæmatology a full-time staff of at least a senior hæmatologist with two medically qualified hæmatologists and a number of highly-trained technicians.

The data reviewed above indicate that, until the Brisbane General Hospital has a staff of this type, that section of the hospital work will not be done as the hospital pathologists and physicians would wish, nor will the invaluable ward contact with patients be initiated and maintained. This mention of ward contact suggests thoughts of the clinical function of the hospital pathologist. In England there is a strong feeling that senior hospital clinical pathologists should be men with high clinical ability. A number hold a Fellowship or at least a Membership of the Royal College of Physicians, or a degree of doctor of medicine, as well as their special qualifications from long training in clinical pathology. Such men are capable of taking charge of hæmatological clinical units and of giving help at bedside consultations in unusual cases.

Men with double training are scarce everywhere, and presumably very rare in Australia; but if such a man was available, a reasoned view of the future needs of medicine in Queensland would amply justify providing the conditions necessary to secure him—staff, opportunity and leisure for the study of unusual cases, equipment, accommodation and salary.

Some years have elapsed since the need for efficient hæmatological units in large English and American hospitals was recognized and implemented. Have we in Brisbane learnt enough from the history of this development, and have we learnt quickly enough?

Conclusion.

After this expedition we ended our Rip Van Winkle journey by a visit to Jackson's beautiful home, where we discussed many things. His eyes took on a mischievous twinkle when I reached down the "*Religio Medici*" from his library shelf and pointed out the gem: "He who discommendeth others obliquely commendeth himself. He who whispers their infirmities proclaims his own Exemption from them, and consequently says, I am not as this Publican." Eventually the evening came to an end—as all good evenings do—and I like to think I left Jackson as on the last night in 1903 when I saw him there, beaming at his happy guests dancing to his expert handling of an excellent player-piano, and finally bidding us a warm-hearted "good night" from the doorstep.

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TUBERCULOSIS MORTALITY IN AUSTRALIA, 1908 TO 1945.

By H. O. LANCASTER,

School of Public Health and Tropical Medicine, Sydney.

IN this paper, one of a series dealing with mortality in Australia, I shall discuss the mortality from tuberculosis (all forms), leaving a detailed analysis by form and site of the disease to later papers. I shall also defer consideration of the disease in childhood.

SOURCES OF DATA.

The deaths from tuberculosis are listed in various tables in *Demography*, the annual bulletin of the Bureau of Census and Statistics, Canberra. For an account of the deaths from all forms of tuberculosis, the most convenient table is that entitled "Deaths from Tubercular Diseases: Length of Residence in Australia in Age-groups, Australia". For the years prior to Federation, it is difficult to tabulate the tuberculosis deaths from each Colony for each year and so obtain a total for all Australia and then sum these into deaths by decades. Therefore, I shall use the rates for the Colony of New South Wales for the three years about the Census of 1891—that is, the years 1890 to 1892—as representative of the rates for all Australia. I shall further assume that these rates are representative of the whole decade 1891 to 1900. It will be found that this approximation is unlikely to affect the conclusions drawn from the data when they are analysed by the "generation method" to be described later.

A Note on the Classification of the Causes of Death by the Statistical Offices.

The general practices of the statistical offices have been described elsewhere (Lancaster and Willcocks, 1950). It suffices to note here that if two causes of death are entered on the death certificate, then only one may be selected for the purposes of classification. A coding manual was used to guide the statistician in this selection. It may be said that "tuberculosis" had a high priority and was to be preferred to almost any other form or cause of death appearing on the death certificate, with the exceptions of "cancer", "violence" and certain rare infective conditions. So that, if the diagnosis "tuberculosis" appeared on the certificate, the death would, with almost complete certainty,

TABLE I.

Tuberculosis Mortality in Australia, 1908–1945: Deaths from Tuberculosis (All Forms) by Age and Sex for Australia for Five Periods in the Years 1908–1945.

Age Group. (Years.)	Males.					Females.				
	1908–1910.	1911–1920.	1921–1930.	1931–1940.	1941–1945.	1908–1910.	1911–1920.	1921–1930.	1931–1940.	1941–1945.
0–	413	1101	768	441	206	317	928	663	365	150
5–	98	291	216	129	47	87	285	196	130	59
10–	101	252	194	128	39	129	352	254	153	58
15–	285	741	626	360	131	487	1240	1191	713	223
20–	509	1692	1365	853	284	739	2263	2103	1515	509
25–	1235	4531	4012	2452	817	1385	4357	4095	3136	1230
35–	1319	4497	4477	3303	1343	923	2888	2694	2190	909
45–	1206	4127	4090	3921	1908	530	1676	1603	1305	595
55–	665	2596	3054	3237	1913	260	919	1015	966	465
65–	335	1004	1380	1782	1167	153	435	526	645	370
75–	78	210	260	422	315	37	115	146	231	151
Unclassified	10	27	23	6	3	3	3	1	2	0
Total ..	6254	21069	20465	17034	8173	5050	15461	14487	11351	4719

be referred to one of the rubrics of the International List dealing with tuberculosis. However, possibly there were some losses to "violence" for the earlier ages at death and to "cancer" in the later ages. Another source of loss might be the failure of the certifying medical officer to give the diagnosis "tuberculosis" on social grounds. We have no means of assessing the numerical importance of any of these sources of loss, since there are no Australian data which would help us in coming to a conclusion. It may be argued, on the other hand, that some of the cases assigned to "tuberculosis" by the coding rules were not really deaths from tuberculosis; for instance, had a diabetic died in coma and "tuberculosis" been mentioned on the death certificate, the death would have been referred, according to the coding rules, to one of the tuberculosis rubrics, since these rules gave an unvarying preference to "tuberculosis" over "diabetes". Another disturbing factor was the increased interest in tuberculosis and the widespread use of mass radiography in the later years, especially in connexion with the "call-up" for the armed services in the recent war. Notwithstanding all these factors, it seems fair to conclude that the official statistics do give an approximate answer to the question: "How many persons does tuberculosis kill?" Now that a new method of assessment of the cause of death is to be instituted by international agreement, it is probable that future tuberculosis mortality statistics will not be comparable with those of the period studied.

TOTAL DEATHS FROM TUBERCULOSIS.

In Table I are given the deaths from all forms of tuberculosis for all Australia by age and sex for each of the periods studied. The five-year age-groups have been retained up to the age of twenty-five years, since in the early years of life the mortality rates for tuberculosis change rapidly with increasing age. After the age of twenty-five years, ten-year age-groups have been used up to the age of seventy-five years. All the deaths of persons over this age have been combined into a group as "seventy-five years and over".

Table I is of no great importance from a comparative point of view, but serves as a check on all the other computations and on the analysis of the tuberculosis deaths into occurrence by site. To display tuberculosis as an important caues of death, I have computed for certain broad age groups the percentages of all deaths that are due to tuberculosis. In Table II are the number of deaths in each of the periods studied for certain broad age-groups.

These broad age-groups are defined as nil to four years last birthday, five to fourteen years, fifteen to forty-four years, forty-five to seventy-four years, and seventy-five years and over. These ages correspond roughly to the times of early childhood, later childhood, early adult life, later adult life and old age respectively. In Table III is set out the percentage of the total mortality in each of these broad age-groups that is due to tuberculosis for each of the five periods. When this has been done, it is obvious that tuberculosis is revealed as a very important cause of death indeed. Thus we find that, in the period 1908 to 1910, over a quarter of all female deaths in the age-group fifteen to forty-four years were due to tuberculosis, and that the proportion of male deaths at the same ages was almost as high. We may also use this percentage in each age-group as an indication whether the mortality from tuberculosis has fallen as rapidly as that from all other causes. In general we notice that the mortality from tuberculosis is decreasing more rapidly than that from all other causes, since the percentage of the mortality due to tuberculosis is decreasing in practically every age-group. In fact, the only exceptions to be made to the general statement are the single trivial rise in the 1921 to 1930 period in the fifteen to forty-four years age-group, as compared with the previous period, 1911 to 1920. Comparisons are made by age for any given period with much less safety, since the mortality from all other causes changes so much with age; for example, the fact that tuberculosis was a very important cause of child mortality in the years 1908 to 1910 is rather obscured. At the other end of life, the mortality from all other causes is so high that the per-

TABLE II.

Tuberculosis Mortality in Australia, 1908–1945: Deaths from Tuberculosis (All Forms) in Certain Broad Age Groups for Australia.

Age Group. (Years.)	Males.					Females.				
	1908–1910.	1911–1920.	1921–1930.	1931–1940	1941–1945	1908–1910.	1911–1920.	1921–1930.	1931–1940.	1941–1945.
0 to 4 ..	413	1101	768	441	206	317	928	663	365	150
5 to 14 ..	199	543	410	257	86	216	637	450	283	117
15 to 44 ..	3348	11461	10480	6968	2575	3534	10748	10083	7554	2871
45 to 74 ..	2206	7727	8524	8940	4988	943	3030	3144	2916	1430
75–	78	210	260	422	315	37	115	146	231	151
Unclassified	10	27	23	6	3	3	3	1	2	0
All ages ..	6254	21069	20465	17034	8173	5050	15461	14487	11351	4719

TABLE III.

Tuberculosis Mortality in Australia, 1908–1945. The Importance of Tuberculosis as a Leading Cause of Death. (The Proportion of Tuberculosis Deaths, All Forms, to Deaths from All Causes is Given as a Percentage.)

Age Group. (Years.)	Males.					Females.				
	1908–1910.	1911–1920.	1921–1930.	1931–1940.	1941–1945.	1908–1910.	1911–1920.	1921–1930.	1931–1940.	1941–1945.
0–	2·21	1·63	1·38	1·24	1·11	2·12	1·74	1·54	1·33	1·05
5 to 14 ..	7·92	5·49	4·32	3·02	2·48	9·95	7·47	6·11	4·74	5·04
15 to 44 ..	21·89	19·21	19·50	14·26	13·95	26·43	21·80	21·36	18·15	16·28
45 to 74 ..	7·69	6·54	5·90	5·02	4·89	5·26	4·15	3·30	2·34	1·95
75–	0·60	0·42	0·48	0·51	0·57	0·29	0·28	0·29	0·30	0·27
All ages ..	7·99	6·91	6·44	4·82	4·13	8·72	6·84	5·94	4·10	2·89

centage may lead to a gross under-estimate of the importance of tuberculosis at these later ages.

THE AGE-SPECIFIC DEATH RATES FROM TUBERCULOSIS.

The age-specific death rates from tuberculosis are set out in Table IV. They have been obtained by dividing the tuberculosis deaths by suitable estimates of the total number of years lived by individual persons of the same sex and age-group over the given period. These total numbers of years lived have been obtained by a summation of the estimates of the mean populations of Australia of that age-group for each year of the period considered.

The age-specific death rates may be compared in three different ways, as was pointed out by M. Greenwood (1936) in a discussion of the death rates from all causes in England and Wales.

The First Method.

The first method is that commonly adopted—namely, for a given calendar year or period, such as 1911 to 1920, the age-specific rates are compared as in Table IV or Figure I. By this method, we notice that in the period 1911 to 1920 for males there is initially a high death rate for tuberculosis of 387 *per annum* per million at ages nil to four years last birthday. The mortality rate then falls to a minimum of 106 at ages ten to fourteen years, and then rises steadily to a maximum at ages fifty-five to sixty-four years of 1612 *per annum* per million. The course of the female mortality rate is somewhat different. After beginning with 332 *per annum* per million at ages nil to four years, the death rate reaches a minimum in the five to nine years age-group and then rapidly rises to attain a maximum of 1074 in the twenty-five to thirty-four years age-group. From the age of thirty-five years the rate then steadily declines to a rate of 358 *per annum* per million for females over the age of seventy-five years. Such an analysis makes the implicit assumption that the rates at ages twenty-five to thirty-four years, for example, are comparable with those at ages thirty-five to forty-four years and so on—that is, that it is possible to imagine a group of persons beginning life at the rates of the nil to four years age-group of the actual population and passing through life with the mortality from tuberculosis or other causes of death equal at each age to that of the actual population. This is, of course, the basic assumption for the construction of a life table. We shall see later that this method may give quite a fallacious idea as to the age at which the greatest mortality from tuberculosis is occurring in any group (or cohort) of persons followed throughout life. This objection applies equally strongly to arguing from the results of Mantoux reaction surveys, morbidity data *et cetera* collected in a short period of time to an estimate of what would happen to a group of persons followed throughout their life. It must then be years before any such survey in Australia or elsewhere can give an answer to the question of what is the experience of a group or cohort of persons passing throughout life with regard to immunity, as measured by the Mantoux reaction, or morbidity, although the data are available with regard to mortality.

The Second Method.

A second method is to study the mortality at a given age for each of a number of periods. Whereas the first method would read Table IV by columns, the second would read it by rows. When this is done, it is noted that the rates at each age have fallen for both sexes with the exception of males over seventy-five years of age. The reduction in the mortality rates has been most noteworthy under the age of twenty years in both sexes. Detailed consideration of the mortality under fifteen years of age will be referred to in a later article, in which will be given the comparison of the rates at each age for each sex for the different periods graphically.

The Third Method.

The third method of interpreting the rates of Table IV is the "generation method", which will be discussed in a later paragraph.

TABLE IV.

Tuberculosis Mortality in Australia, 1908–1945: Mortality from Tuberculosis (All Forms) Expressed as Rates per Million of Persons at Risk.

Age Group. (Years.)	Males.					Females.				
	1908–1910.	1911–1920.	1921–1930.	1931–1940.	1941–1945.	1908–1910.	1911–1920.	1921–1930.	1931–1940.	1941–1945.
0–	533	387	244	155	126	418	332	217	133	95
5–	151	111	70	43	34	134	109	65	44	44
10–	165	106	65	40	27	211	148	87	50	42
15–	429	317	226	115	82	733	537	444	235	145
20–	785	749	531	282	181	1139	989	859	517	332
25–	1150[1]	1104	844	447	277	1384[1]	1074	866	607	424
35–	1548	1365[1]	1056	707	527	1215	935[1]	667	469	374
45–	1720	1587	1337[1]	982	877	957	745	562[1]	338	267
55–	1988	1612	1335	1225[1]	1175	939	664	488	372[1]	284
65–	1704	1276	1138	1089	1325[1]	857	605	478	390	384[1]
75+	861	650	648	677	799	468	358	336	336	319
All ages ..	939	837	670	497	449	817	639	494	340	262

[1] Rates taken to be appropriate for the cohort of 1876.

The Influence of Sex.

The effect of sex can be discussed by comparing the rates of the two sexes at each age and for each period. This is most simply done by expressing the male mortality as a fraction of the corresponding female mortality. This has been done in Table V, in which the ratio (male mortality) : (female mortality) for each age group in each period is set out. If we choose for the comparison any one period, say 1931 to 1940, we note that in infancy the ratio is 1·17, so that the male mortality rates are some 17% higher than the female rates. This ratio falls to unity approximately in the age-group five to nine years and then reaches a minimum at ages fifteen to nineteen years. There is then a steady rise with a maximum of approximately 3·3 for the ratio in the age-group fifty-five to sixty-four years. The full explanation of these changes in the relative magnitudes of the mortality rates in the two sexes is not known.

TABLE V.

Tuberculosis Mortality in Australia, 1908–1945: The Ratio of the Male Death Rates from Tuberculosis (All Forms) to the Corresponding Female Rates.

Age Group. (Years.)	Period.				
	1908–1910.	1911–1920.	1921–1930.	1931–1940.	1941–1945.
0–	1·28	1·17	1·12	1·17	1·33
5–	1·13	1·02	1·08	0·98	0·77
10–	0·78	0·72	0·75	0·80	0·64
15–	0·59	0·59	0·51	0·50	0·57
20–	0·69	0·76	0·62	0·55	0·55
25–	0·83	1·03	0·97	0·74	0·65
35–	1·27	1·46	1·58	1·51	1·41
45–	1·80	2·13	2·38	2·91	3·28
55–	2·12	2·43	2·74	3·29	4·14
65–	1·99	2·11	2·38	2·79	3·45
75+	1·84	1·82	1·93	2·01	2·50

It may be remarked in passing that the female death-rates are more favourable than the male for most diseases in infancy and early childhood. However, at puberty it is evident that there must be some unfavourable factors acting on the female. Child-bearing has often been invoked as a possible explanation, but we see that the female rates are already less favourable than the male rates at ages ten to fourteen years. This would also appear to exclude such factors as anæmia arising from excessive menstruation. Social factors have also been brought forward to explain this fall in the ratio. The reader is referred to a paper of A. Bradford Hill (1936) for a discussion of many of the theories. In later life the increase in the ratio is believed to be due to occupational factors.

The Crude and Standardized Mortality Rates.

It is usual to give the crude death rates from disease as a measure of the load that the population is carrying at any time. In Table VI we give the crude rates of death from tuberculosis for each of the periods studied. It is seen that there has been a fall in the crude rates to some 50% of the 1908 to 1910 rates for males and some 30% for females in the years 1941 to 1945. However, for most diseases it is desirable to remove the effect of age from the comparisons to be made, and so in Table VI is given the mortality standardized onto two standard populations, firstly the actual population of England and Wales enumerated at the Census of 1901, and secondly, the life table populations constructed from the data of the 1933 Census of the Commonwealth of Australia. In either case, standardization shows that the fall in the crude rates cannot be due merely to changes in the age distribution of the population, and that the percentage falls in the rates are of the same order of magnitude whether we use the crude or the standardized rates for comparison. Standardization estimates what number of deaths might be expected in the standard population were it submitted to the same rates of mortality as are observed in the actual population being studied. The process of standardization alternatively may be regarded as the computation of a weighted mean of the age-specific rates. The tuberculosis mortality is spread over such a wide span of the life cycle that standardization is not of the crucial importance here that it is in the case of the comparison of cancer rates, for cancer is a disease restricted very largely to the later years of life. For tuberculosis, the mortality rates at early ages have decreased more than those at the later ages, so that we find that the rates standardized onto the England and Wales population of 1901 show a greater relative decline than the rates standardized onto the life-table population, since in the former case the young persons are more heavily represented.

A Fallacy Arising from Interpreting the Age-specific Rates for a Calendar Period as the Experience of a Group of Persons Followed Throughout Life.

I shall now discuss in greater detail some difficulties in interpretation of the age-specific rates by the first method described above. In the formation of a life table one assumes that a group of persons beginning life, say 100,000 males, can be imagined to be followed throughout life submitted to the same rates of mortality as hold in some actual observed population—for example, the population of Australia in the three years 1932 to 1934. Essentially the same sort of reasoning lies behind the comparison of the age-specific death rates at a point of time or for a calendar period such as 1911 to 1920. We assume that it is fair to compare the mortality of persons aged twenty years with that of persons aged forty years, although up to the age of ten years these two groups of persons may have been submitted to very different experiences of infection. Thus it is customary to consider such a diagram as Figure I, in which the rates in the period 1911 to 1920 are compared with the rates in the period 1941 to 1945. It is noted that in the period 1911 to 1920 the mortality was spread out over a broad range of years, whereas in the later period—1941 to 1945—the mortality was at its highest in the later years of life. This has been explained by many as an indication that the high mortality in the later age-groups is due to the infection with the tubercle bacillus being deferred to a later age in more recent times; they argue that in the earlier times the brunt of infection was borne in childhood and the resulting survivors were largely immune persons with latent infections, and so the "excess" mortality in the more recent times at the higher ages was the result of a relatively "unsalted" population being

TABLE VI.

Tuberculosis Mortality in Australia, 1908–1945: The Crude and Standardized Death Rates from Tuberculosis (All Forms) for Australia as Rates per Million per Annum.

Period.	Crude Rates.		Rates Standardized for Age: Standard Population Used.			
			England and Wales, 1901.		Australian Life Table Population of 1933.	
	Male.	Female.	Male.	Female.	Male.	Female.
1908–1910 ..	939	817	912	823	1146	858
1911–1920 ..	837	639	795	639	967	657
1921–1930 ..	670	494	617	492	777	506
1931–1940 ..	497	340	422	327	578	349
1941–1945 ..	449	262	351	242	521	270

attacked by tuberculosis. Such considerations are easily seen to be fallacious if we note that, although in Australia the mortality in the period 1941 to 1945 was higher in the age-group fifty-five to sixty-four years than in any other age-group for males, yet it is lower than the corresponding group for the period 1911 to 1920, and indeed for all the four earlier periods. It is apparent that part of the misconception arises because the ratio of death rates at the different ages is compared for the different periods; that is, the shape of the graphs is compared rather than their actual height. We can discuss this problem adequately only with the aid of the "generation" method, which is the third method referred to in a previous paragraph.

The Generation Method of Analysing the Age-Specific Rates.

W. Hampton Frost (1939) gives a clear discussion of the "generation" method applied to the rates for Massachusetts. He was fortunate in having a body of data extending over a considerable period of time from 1870 to 1930. Our Australian data, on the other hand, have a duration of only some forty years. Therefore the data for New South Wales for the three years about the census of 1891, the years 1890 to 1892, have been used as if they were typical of all Australia and on the assumption that the rates did not change greatly over the decade 1891 to 1900. Thus these data have been taken to be typical of Australia over the decade. It will be found that this approximation, and a similar approximation with regard to the 1941-1945 rates of Australia which are assumed to be representative of the decade 1941 to 1950, do not weaken the general argument of this section. The general conclusions that will be drawn are as follows. (i) The present era is one of declining death rates from tuberculosis. (ii) This decline leads to an appearance that the mortality from tuberculosis is postponed from the earlier years of life to the later years of life if the mortality rates, observed in a calendar period, are read as being the rates of a group of persons followed throughout life. (iii) This appearance is an artefact; if one attempts to follow an actual group of persons throughout life the greatest mortality occurs in females at the age of thirty years and also in males at this age, although the evidence is somewhat weaker.

The term "cohort" has been introduced into vital statistics merely as a collective noun, in the sense of a group or collection of persons. Certain other almost synonymous descriptive terms such as "group" tend to become overworked. Other words such as "legion" have associations that make them unsuitable for the purpose, while the use of "generation" itself would lead to confusion, since we may desire to select groups of persons not necessarily born so far apart as a generation.

The generation method will now be discussed in some detail. A similar description is available in Greenwood's account of the English death rates (1936). Other descriptions are available in the papers of Frost (1939), of Derrick (1927), of Kermack, McKendrick and McKinlay (1934), and of Pollard (1949). For medical readers the account of Frost will be found the easiest. Let us imagine that we have the mortality rates for males for each year of life in each calendar year from 1896. Then we may study the death rates in 1896 for children under the age of one year; these rates will represent the death rates of children born on the first day of the year. Similarly in 1897 the death rates of children aged one year last birthday will represent the death rate of the cohort born at the beginning of 1896, which we shall continue to call the cohort of 1896. If now we desire to find the death rate of our cohort at age x years, then we look for the death rate of children aged x years last birthday in the calendar year $1896 + x$. Thus we shall be able to follow the mortality of the cohort throughout life until we come to the current year. However, it would be found even in the case of large populations that a dissection of the death rates each year by single years of age, by sex and by cause of death would result in rather large sampling errors, as measured by the coefficient of variation, if we desired to compare the experience of cohorts of neighbouring years. Therefore the unit of time chosen has been not the year, but the

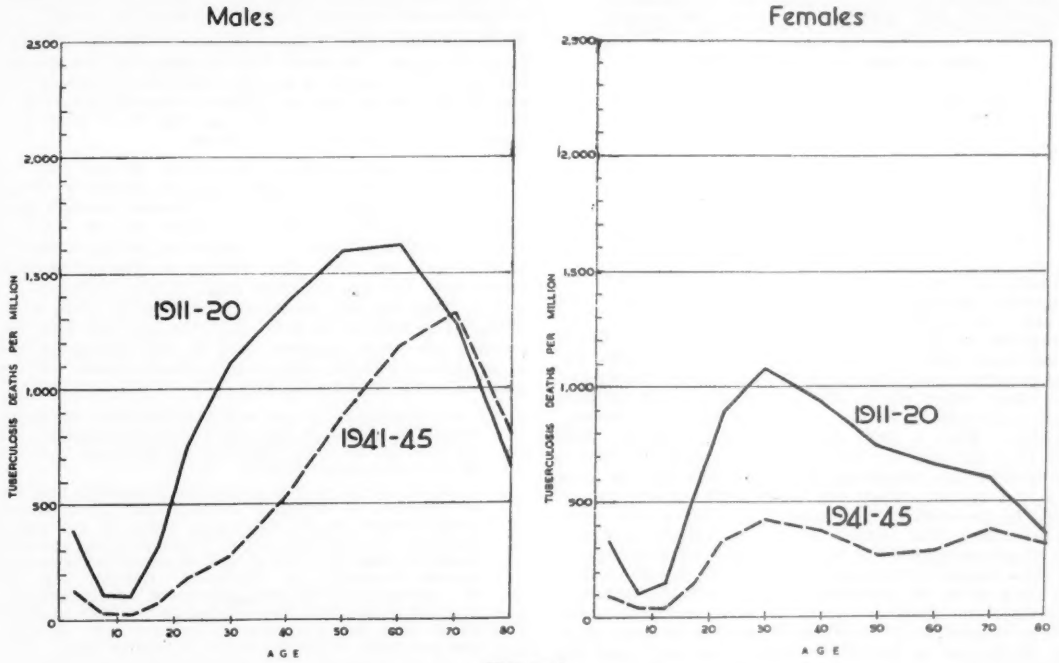

FIGURE I.

The death rates from tuberculosis (all forms) for Australia for two periods of time, 1911 to 1920, and 1941 to 1945. Arithmetic grid.

decade. We can now follow such a cohort as that of males born in the decade 1891 to 1900, which in the tables and diagrams has been termed the cohort of 1896. This cohort will be aged five to fourteen years in the decade 1901 to 1910. Thus the mortality rates of 1901 to 1910 for the corresponding age group five to fourteen years have been assigned to this cohort, as also the rates for ages fifteen to twenty-four years in the 1911 to 1920 period and so on. It is found that this procedure is equivalent to reading Table IV by diagonals; for example, the mortality rates of the cohort of 1876 are given by the figures in the diagonal marked by [1]. The relations at ages under twenty-five years are obscured in Table IV, since the mortality rates at earlier ages are given by five-yearly age groups. We have therefore had to combine the four five-year groups five to nine, ten to fourteen, fifteen to nineteen and twenty to twenty-four into two age groups—five to fourteen years and fifteen to twenty-four years.

TABLE VII.

Tuberculosis Mortality in Australia, 1908–1945: The Mortality Rates for Certain Age Groups for the Colony of New South Wales in the Years 1890 to 1892 from Tuberculosis, Scrofula and Acute Hydrocephalus Combined, Used as an Approximation to the Corresponding Rates for All Australia at That Time.

(Rates per Million per Annum at Risk.)

Age Group. (Years.)	Males.	Females.
0—	1924	1639
5—	173	161
15—	904	864
25—	1903	1755

In Table VIII are set out the mortality rates that we have thought appropriate to each of the cohorts. The data are displayed graphically in Figure II. The interpretation of the female death rates is definite. There is a maximum for the tuberculosis mortality rates for each of the first four cohorts in the age group twenty-five to thirty-four years, but in the cohort of 1906 it appears that the maximum is in the age group fifteen to twenty-four years. With a finer dissection of the rates, with unit of time for the age groups of five years, we should expect to find the maximum mortality rate in any case close to the age of twenty-five years. The maximum as observed in the cohorts is sharper than that of the rates in the calendar period method outlined previously, although in the diagrams this effect is exaggerated by a change of scale. The results of the application of the cohort method to the male rates of mortality are less definite, although it appears in the diagram that there is a maximum in the age group twenty-five to thirty-four years or in some cases in the thirty-five to forty-four years. In any case the maximum in the later age groups, observed in a single period by the first method, does not appear so definitely. In fact, if the rates for the period from 1941 to 1945 were excluded, then our conclusions would be similar to those drawn in the case of the females. It is to be noticed that the disturbing effect of the 1941 to 1945 rates makes itself felt at different ages in the different cohorts. I believe that there must be some disturbing factor in the case of the male rates, either due to the war and the consequent increased radiographic diagnosis of tuberculosis in recruits to the armed forces, or to increased awareness of the disease in the susceptible occupations. No great error will result if we suggest that there is a maximum in the male rates at the age of approximately thirty-five years.

That such findings may occur in other countries with a declining death rate from tuberculosis can be illustrated by the conclusions of W. H. Frost (1939) on the Massachusetts data. In the following passage he is commenting on the analysis of the mortality rates by the first method above—that is, the comparison of the rates at different ages at a point of time:

> (1) At every age the mortality is lower in the later period.
>
> (2) In each period age selection is generally similar: mortality is high in infancy, declining in childhood, rising in adolescence to a higher level in adult life.
>
> (3) In the later period (1930) the highest rate of mortality comes at the age of fifty to sixty, whereas formerly it was at the age of twenty to forty.

It is apparent that these conclusions fit the Australian data for males. We have already noted the interpretation put upon this change in the age selection by some authors, and Frost admits that he originally shared the view that the change in age selection was due to a loss of the immunizing infections of childhood.

However, examination of the rates by the generation method has shown this conclusion to be fallacious. Persons aged, say, sixty years in 1935 have passed through greater mortality risks in the past, as shown by the very high rate for children aged ten years of a period forty years earlier—that is, 1895. The cohort or generation method shows that the age selection may be very different for a cohort from the age selection at any given time such as 1935. It is also to be noted that the age selection in the cohorts has been uniform—that is, the maximum occurs at approximately the same age in each cohort. We may quote Frost's final conclusions:

> (1) Constancy of age selection (*relative* mortality at successive ages) in successive cohorts suggests rather constant physiological changes in resistance (with age) as the controlling factor.
>
> (2) If, as we may suppose, the frequency and extent of exposure to infection in early life have decreased progressively decade by decade, there is no indication that this has had the effect of exaggerating the risk of death in adult life due to lack of opportunity to acquire specific immunity in childhood. Present day "peak" of mortality does not represent postponement of maximum risk to a later period, but rather would seem to indicate that the present high rates in old age are the residuals of higher rates in earlier life.

There seems little to add to Frost's conclusions on the Massachusetts data. The contrary conclusion that the protection of the young endangers their life at a later period of life would appear to be founded on an analogy with the doctrine of the "survival of the fittest". Frost concluded that, rather than there being a survival of the

TABLE VIII.

Tuberculosis Mortality in Australia, 1908–1945: The Mortality Rates from Tuberculosis (All Forms) for Australia Rearranged to Give the Presumed Mortality Experience of Successive Generations (or Cohorts). The Rates of Mortality at Each Age are Given per Annum per Million Persons at Risk.

Age Group. (Years.)	Males: Cohort Born in the Years Stated.					Females: Cohort Born in the Years Stated.				
	1866.	1876.	1886.	1896.	1906.	1866.	1876.	1886.	1896.	1906.
5—	—	—	173	157	108	—	—	161	172	128
15—	—	904	582	529	373	—	864	934	762	642
25—	1903	1150	1104	844	447	1755	1384	1074	866	607
35—	1548	1365	1056	707	527[1]	1215	935	667	469	374[1]
45—	1587	1337	982	877[1]	—	745	562	338	267[1]	—
55—	1335	1225	1175[1]	—	—	488	372	284[1]	—	—
65—	1089	1325[1]	—	—	—	390	384[1]	—	—	—
75+	799[1]	—	—	—	—	319[1]	—	—	—	—

[1] The rates for the years 1941 to 1945.

fittest, if the rates are high in childhood, the survivors are so damaged that they will have a higher mortality rate than would have been the case with lighter rates of infection in childhood. The statistics then do not controvert the thesis that it is good to avoid infection with the tubercle bacillus in childhood.

A Note on the Method of Generations or Cohorts Applied to a Study of Death Rates in General.

It is possible to determine an "order of dying out"; that is, one can watch 1000 animals from birth, or the hatching of the eggs until all the birds are dead, recording the time of death of each individual. The number of survivors can then be computed at any time and a table of survivorship formed. This procedure is quite impracticable for man, so that various devices have been adopted to form a life table. It is assumed that an imaginary group of, say, 100,000 persons are submitted at every age to the same rates of mortality as exist in some actual population —for example, Australia, 1932 to 1934. The expected survivors can be calculated for each age until, at the age of approximately one hundred years, all are imagined to have died. It has always been recognized by actuaries that except in a time of stationary mortality, as in England and Wales in the middle half of the last century, these life tables did not represent the dying off of any actual population, and that in general the method under-estimated the survivors at every age since the death rates were falling. However, it has never seemed fit to them that they should assess future mortality too optimistically when constructing the monetary tables based on the life tables. Modern interest in the application of the generation method appears to have begun with the article of V. P. A. Derrick (1927), who used English data. A. H. Pollard (1949) has recently applied the method to the forecasting of the Australian mortality. Authoritative work has been done by the Scandinavian and Italian actuaries, but I do not think that justice can be done to them here, except to mention that it seems that Andvord (1930), cited by Frost (1939), was the first worker to apply the generation method consciously to the interpretation of the tuberculosis rates. However, some notice must be taken of the study of Sir Robert Philip on the decline in the rates in Scotland (Philip, 1924). Philip noted that in general the rates first fell in the young and then later the decline passed down the age groups. He noted that the larger reductions were to be found at the earlier ages and the smaller reductions at the later ages, and he ascribed the change in the graphs of the age mortality at ages between 1871 and 1921 to a reduction at the earlier ages without a correspondingly large fall in the mortality rates at the advanced ages. He noted that "there is an apparent postponement of the age at death". A casual inspection of the age-specific mortality rates set out as an appendix to his paper suggests that the generation method would apply very well to his data. M. Greenwood (1927), in a discussion on the paper of V. P. A. Derrick (1927), is reported as follows:

> For example, he (the speaker) had read a paper by Sir Robert Philip, who attached very great importance to the shifting of the age incidence, because he argued that, first of all, the effect of modern treatment was to help the young, and that the change in the death-rate at the later ages would begin many years after the death rate at the earlier ages had been affected. It seemed to him (the speaker) that almost any hypothesis could be made to cover those facts.

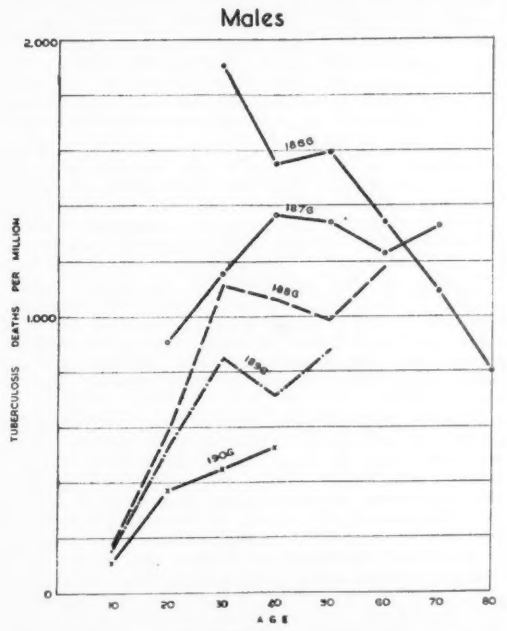

FIGURE IIA.

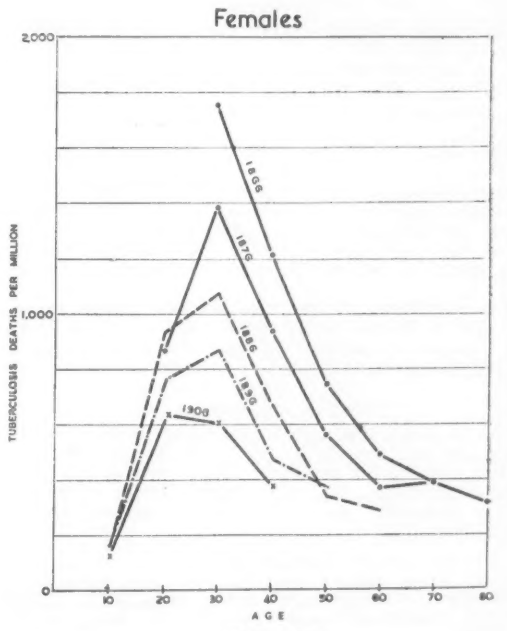

FIGURE IIB.

The death rates from tuberculosis (all forms) studied by the generation (or cohort) method. Arithmetic grid.

Later he was reported as follows:

> For the present, however, he ventured to feel that the method of extrapolation proposed was quite as romantic as any of the extrapolations that medical persons like himself had attempted.

It is clear that the idea of the generation method was not received into medical statistics without opposition. However, the work of Kermack, McKendrick and McKinlay (1934*a* and *b*) on total mortality reintroduced the topic into medical literature. They found that the mortality experience could be expressed as a product of two factors, one depending on the year of birth (and naturally the country) of the cohort and the other on the age of the members of the cohort. In general it may be stated that for total mortality, a cohort having a favourable experience at the early ages will also have a favourable experience throughout life. This is an important conclusion, for it contradicts the thesis so often brought forward that

hygienic measures actually weaken the population in the generation to which they are applied, and that if many infantile deaths are avoided the generation will suffer greater mortality in the future. We note that this argument is applied with even greater force to the case of mortality from tuberculosis. It is evident that the method of generations or cohorts must be applied to the study of any of the chronic diseases of long standing if fallacious conclusions are not to be drawn from official statistics and other forms of data.

Forecasting the Australian Experience for Tuberculosis.

If the graphs of the female rates are used, it seems obvious that we may expect further falls in the female death rates. The equivocal results caused in the male rates by the war years 1941 to 1945 must make us cautious in attempting to forecast a further fall in them. But we may notice that Pollard (1949) gives as a forecast of the rates for the five-yearly age groups centred on ages 17·5, 22·5, 27·5, 32·5, 37·5, 42·5 and 47·5 years in 1960, figures of 30, 60, 130, 180, 260, 400, and 600 respectively per million per annum. (I have read these results from his graph.) These are all substantial reductions on the current rates of mortality.

Summary.

1. The deaths from tuberculosis (all forms) in Australia have been summarized for the years 1908 to 1945, and the trends in mortality studied.

2. Over this period there has been a remarkable decline in the death rates, although this decline began before the year 1908.

3. The death rates have been analysed by several methods.

4. The generation method reveals that the so-called postponement of death to the later years is an artefact, and that the high rates in later life represent the residual effect of high infection rate in the past as evidenced by a high mortality rate of the same cohorts at the earlier ages.

5. The experience of Australia in this respect is not unlike that of Massachusetts as discussed by W. H. Frost.

6. A forecast is quoted. In any case we may expect a further decline in the rates.

Acknowledgements.

I wish to thank Professor E. Ford and Professor A. H. Baldwin, of the School of Public Health and Tropical Medicine, Sydney, for reading a rough draft of the paper and for suggestions. Mr. D. W. Davies of the school has made the diagrams. Mr. W. J. Willcocks has supplied the data of Table VII.

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SALT DEPLETION AND MERCURY POISONING: POTENTIAL DANGERS IN THE TREATMENT OF THE FAILING HEART.

By K. V. Sanderson,
Repatriation General Hospital, Springbank, South Australia.

A GREAT deal has been written about the value of restricting the intake of salt in the treatment of congestive cardiac failure. Sodium chloride is retained in considerable amounts in œdema fluid, and although the mechanism of retention has not yet been fully explained, it is a logical procedure to restrict the intake of salt to a minimum and thus prevent the formation of œdema or hasten its removal. Little mention has been made of any dangers in this treatment until the recent papers of Soloff and Zatuchni (1949) and of Schroeder (1949). In these papers it is shown that the syndrome of salt depletion may complicate this form of treatment and cause death if the condition is not promptly recognized and treated.

It is not very difficult to produce a diet in which the salt content is 0·5 gramme a day, provided that salt-free bread is obtainable. However, such a diet is remarkably tasteless, and although some patients find that potassium chloride or one of the proprietary salt substitutes adds sufficient savour to their food, there are others who beg for the treatment to be stopped. One feels that the syndrome of salt depletion has not been found more frequently because only a minority of patients have sufficient strength of character to maintain the diet over a long period of time. However, as various authors have pointed out, the symptoms of the condition may be confused with those of the primary disease and remain unrecognized.

Organic mercurial compounds are used in congestive cardiac failure as the major diuretic agents. The toxic manifestations of these drugs have been reviewed by DeGraff and Nadler (1942), who emphasize the important role electrolyte depletion plays in producing toxic states. The pharmacological action of mercurial diuretics in the presence of congestive cardiac failure is to stimulate the excretion of sodium salts (Reaser and Burch, 1946). It would therefore seem to be a hazardous measure to limit the intake of salt with a diet of low salt content and at the same time to promote its excretion with a mercurial diuretic. If both treatments are continued effectively for a sufficient length of time, a dangerous state of salt depletion must result. The case reported here illustrates this, and also the danger of mercury poisoning if the diuretic is used when the oliguria of the salt depletion syndrome is established.

Report of a Case.

An ex-serviceman, aged forty-nine years, was admitted to the Repatriation General Hospital, Springbank, on October 21, 1949, complaining of diarrhœa, drowsiness, weakness and shortness of breath, of several weeks' duration. While a prisoner of war in Italy in 1941 he had suffered from rheumatic fever, which was not treated. Since then he had been short of breath, and in 1943 was discharged from the army as medically unfit with the diagnosis of rheumatic carditis. He had been treated at this hospital twice previously, once in June, 1948, for congestive cardiac failure and auricular fibrillation, which responded to rest, digitalis and mersalyl, and again in May, 1949, for congestive cardiac failure, when he was put on a diet of low salt content. During this latter period he collapsed and became cyanosed immediately after an injection of mersalyl, and was considered to be sensitive to the drug.

He was discharged from hospital in August, 1949, and remained on a diet of low salt content at home, and received intravenous injections of two millilitres of "Neptal" once a week from his local doctor. He had remained well on this régime until three weeks before his admission to hospital, when he suffered from nausea, vomiting and diarrhœa for two days, which he attributed to a pie he had eaten. Then he passed a large quantity of "foul-smelling thick urine" not induced by "Neptal". Next his throat became sore and dry. Ten days before his admission to hospital his urine became scanty and foul-smelling, and the injection of "Neptal", given three days before his admission, did not produce the usual diuresis. The day afterwards he had a foul taste in his mouth, and the next day he developed profuse watery diarrhœa, passing eight motions in a few hours, in which he estimated he lost nine pints of fluid. He had been feeling tired and weak for several weeks, with headache, irritability and loss of appetite, all of which were unusual for him. His shortness of breath had been increasing.

Examination showed the patient to be a drowsy, florid-faced, cyanosed man with cold, clammy extremities. He was not in great respiratory distress, but had Cheyne-Stokes breathing. It was obvious that his mental state had changed from a previous cheerful optimism to depression and apathy with a vague sense of impending disaster. His temperature was 102·8° F., his pulse rate was 80 per minute and his respirations numbered 26 per minute. His tongue was dry and his breath smelt stale. His heart was considerably enlarged and fibrillating, with an apical systolic bruit. There was evidence of pulmonary congestion of moderate degree, the liver was enlarged five centimetres below the right costal margin and the jugular veins were distended. The blood pressure was 150 millimetres of mercury (systolic) and 85 millimetres (diastolic). Examination of the urine showed that it was concentrated and contained sufficient albumin to deposit to half the length of the test tube. Microscopic examination revealed a few hyaline casts and leucocytes in each high-power field of the centrifuged deposit.

The following laboratory data were obtained. The blood contained 14·5 grammes of hæmoglobin *per centum*, with 12,000 leucocytes per cubic millimetre, and had a hæmatocrit value of 44%. The concentration of blood urea nitrogen was 34 milligrammes *per centum*, of serum chlorides expressed as sodium chloride 560 milligrammes *per centum*, and of serum sodium 290 milligrammes *per centum* (the normal range by the method used being 325 to 350 milligrammes *per centum*). The Fantus test showed that his urine contained less than one gramme per litre of sodium chloride. The fæces were sterile, and on microscopic examination only amorphous debris was seen. A skiagram of his chest revealed a grossly enlarged heart and congested lung fields, not materially changed since the previous picture three months before. An electrocardiogram revealed auricular fibrillation with a rate of 80 beats a minute.

A diagnosis of salt depletion was made on clinical grounds and confirmed by the laboratory tests enumerated above. He was given salt and water by mouth *ad libitum*, with a daily minimum of 25 grammes of salt. He took what would normally be a nauseatingly salty drink without any apparent discomfort.

For the first four days he was seriously ill. He had oliguria and a positive fluid balance, and passed less than two grammes of sodium chloride in the urine each day. His diarrhœa was partly controlled by bismuth and opium, but he became increasingly short of breath, and on October 26 400 millilitres of blood were removed by venesection. This lessened the dyspnœa and hepatic congestion. After this his general condition improved, his urinary output returned to normal and he began to excrete up to six grammes of sodium chloride a day. The diarrhœa remained a troublesome symptom for over a fortnight. By October 31 his serum sodium level had risen to 320 milligrammes *per centum*; he was feeling well again, but complained of increasing nocturnal dyspnœa. He was again put on a diet of low salt content and given aminophyllin by mouth. This relieved the dyspnœa, but after two weeks he again became depressed and lost his appetite and the urinary chloride content fell to two grammes per litre. The diet of low salt content was then relaxed somewhat, and since then he has remained in moderately good health.

The Natural History of the Salt-Depletion Syndrome.

As far as can be judged from previous reports in the literature and from the application of physiological principles, the present case illustrates the chief features of the evolution of the salt-depletion syndrome. The initial symptoms, which sometimes follow an infection or gastro-intestinal upset, are anorexia, weakness, headache and mental changes in which irritability, depression and later apathy predominate. This is the stage of mild salt depletion and very little change is apparent on physical examination. Diuresis may occur and temporarily restore the isotonicity of the body fluids. The symptoms develop insidiously, and to them are added dryness of the mouth and throat which is not relieved by water, increasing dyspnœa and oliguria. A moderate to severe degree of salt depletion is now present. Examination reveals a flaccid and weakened muscular system, evidence of hæmoconcentration and peripheral vascular failure, and early renal failure as indicated by albuminuria and the rise in the blood urea content. The signs of congestion do not increase greatly. The temperature may be raised. This is the stage at which further injections of mersalyl can cause mercury poisoning, because the kidneys are unable to excrete the diuretic. The resulting colitis and toxic changes in the kidney will add to the gravity of the condition, as the loss of electrolyte from diarrhœa may be very considerable. The end usually comes suddenly, the patient dying either in coma or from vasomotor collapse. Throughout the course of the disease the excretion of sodium chloride is greatly decreased, and in the later stages the serum sodium content becomes progressively lower.

Differential Diagnosis.

Considerable clinical judgement may be required to differentiate this therapeutically induced complication from a natural complication of the disease being treated. The increasing dyspnœa may lead to a diagnosis of increasing cardiac failure and to the even more energetic prescription of dehydration treatment. Cases in which this has occurred have been recorded. Again, in a hypertensive patient the condition could easily be mistaken for terminal renal failure. The peripheral circulatory collapse might suggest myocardial infarction. Pneumonia could be suspected because of the fever, dyspnœa and cyanosis. These errors can be avoided only if a careful clinical assessment is made, particularly of the manner in which the symptoms developed.

Idiosyncrasy to mercury should cause no confusion, as the sudden collapse and cyanosis immediately following the injection are characteristic and sufficiently alarming to be remembered by anyone who has seen them.

When the low-salt régime is used a further possible complication must be borne in mind—that potassium compounds used as substitutes for common salt may cause toxic effects. Keith and Burchell (1949) conclude that potassium intoxication is likely to occur only when there is severe renal insufficiency. There are no characteristic clinical manifestations, although the diagnosis may be suggested by paræsthesiæ in the hands and feet, sudden weakness of the extremities, or a shock-like state. The typical evidence is a raised serum potassium level and a series of changes in the electrocardiogram which Keith and Burchell describe in detail. Sudden death from cardiac arrest is likely to result. Potassium salts should, therefore, be given with caution when the kidney function is impaired.

Lithium poisoning has been the subject of several reports from America (Corcoran *et alii*, 1949; Hanlon *et alii*, 1949), where lithium salts have been marketed as a salt substitute. As far as I know no similar compounds are on the market in Australia. The symptoms are drowsiness, weakness, tremor, neuro-muscular irritability, anorexia and nausea.

Treatment.

All dehydration measures must be stopped, loss of electrolyte and water through vomitus and diarrhœa must be controlled, and sufficient salt and water must be given to replace the body's depleted stocks. Schroeder found a greater loss of salt than water in his cases and advocated treatment with hypertonic saline given intravenously. He used solutions of up to 6% strength. If such treatment is used, expert biochemical control is necessary, and one feels that in an emergency like this the kidney, even when injured, can control the body's electrolytes more

efficiently than a biochemist. It is best to give the salt and water by mouth if the patient can drink. Marriott (1947) has estimated that in moderate to severe depletion a deficit of 0·5 to 0·75 gramme of sodium chloride per kilogram of body weight may exist, and in very severe depletion it may be twice this figure. The dosage of salt is best judged from day to day by the patient's condition. The myocardium, in addition to suffering from the pre-existent disease, probably suffers from the same asthenia as the somatic musculature, and venesection and administration of digitalis may be valuable in restoring compensation.

It should not be inferred that a diet of low salt content or the mercurial diuretics are so dangerous as to be of limited value or fit only to be used under hospital conditions. Used either separately or together, these measures are probably the most potent therapeutic weapons we possess to treat congestive cardiac failure, and are quite suitable for treatment in the home provided the dangers are appreciated. The desire to make these dangers generally known prompted the publication of this paper.

Summary.

1. A case in which salt depletion and mercurial poisoning were produced by treatment with a diet of low salt content and "Neptal" injections is described, together with the biochemical findings and the treatment used to combat the condition.
2. The symptoms of this condition are anorexia, weakness, headache, mental changes, dry mouth, dyspnœa, oliguria, circulatory collapse, coma and death.
3. The differential diagnosis and treatment of the condition are briefly discussed.

Acknowledgements.

The biochemical estimations were performed by Mr. K. Barber, biochemist at the Repatriation General Hospital, Springbank, and I express my gratitude to him. I am indebted to the chairman of the Repatriation Commission for permission to publish this paper and for the use of materials and records.

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Reports of Cases.

A CASE OF THORACOPAGUS TRIBRACHIUS DIPUS.

By R. J. RIDDELL,

From the Department of Pathology, the Women's Hospital, Melbourne.

A SYMMETRICAL double monster was examined at the Women's Hospital, Melbourne, in December, 1946. These cases are of such rarity that a description of the anatomical findings seems justified.

Clinical Record.

Mrs. J., aged thirty years, had three healthy normal children and was again pregnant. Her three previous pregnancies had been normal in every way and labour had not been unduly prolonged. There had been no history of any abnormality, or of twins, in the wife's or husband's families. Both husband and wife were well built and had had no previous illnesses. The wife had a normal sized pelvis, with the following diameters: interspinous, 24 centimetres; intercristal, 27 centimetres; external conjugate, 20 centimetres. The pregnancy had been normal except for some morning sickness in the first three months, and again during the last two weeks. Labour was due to commence on December 2, 1946.

FIGURE IA.
External appearance of the double monster.

Labour commenced on November 8, 1946, and was very prolonged. After sixteen hours an examination showed a head engaged in the pelvis, but a second head was felt in the left iliac fossa and a diagnosis of twin pregnancy was made. Prolapse of the cord occurred; the cord was not pulsating. The cord was pushed back and forceps applied. Traction produced rotation of the blades and a movement of the second head, and it was assumed that the twins had become locked. Persistent traction resulted in the delivery of one head followed by a second lying in the neck of the first. Delivery of a single trunk was completed without incident. A single placenta was delivered intact, and no trauma was evident in the birth canal on inspection. The puerperium was uneventful.

The clinical details were supplied by Dr. John England, who presented the monster to this department for examination.

Anatomical Description.

The body was that of a double-headed female monster (Figures IA and IB). Two normally formed heads and necks were attached to a single trunk. The trunk had a normal arm on each side and a stump representing a third arm on the postero-superior aspect between the two necks. The chest wall appeared normal anteriorly and the nipples were present in the normal situation. Posteriorly the line

of the dorsal process of the vertebræ of the neck was continued downwards in the corresponding vertebral column, which was seen on inspection on each side of the mid-dorsal line extending down to the sacral region. There was a single umbilical cord attached in the normal position, containing a vein and two arteries. Below the level of the umbilicus the body was that of a normally developed female infant.

Body Cavities.

A skin incision was made extending from the base of the rudimentary third limb to the pubis. The skin flaps were retracted and the anterior thoracic wall was examined. The sternum was present in the normal position, but was much wider than normal. Two clavicles were normal in position articulating with the manubrium, but a third clavicle was also seen stretching across from the rudimentary third limb. The normal number of ribs were present on either side. The anterior wall of the chest was now removed by section through the rib cartilages. The thoracic cavity was seen to contain a double set of organs (Figure II). Two thymus glands were situated in the upper part of the mediastinum on each side, and a large single pericardial cavity occupied a central position in the lower part of the mediastinum. The pericardium contained two hearts. Normal pleural cavities were present on each side containing well-developed lungs. The two medial lungs were of small size, but of normal morphology. They

FIGURE IB.

External appearance of the double monster.

were compressed by a large hernial sac extending from the diaphragm upwards behind the common pericardial sac. The two lateral lungs were rather large and also of normal morphology, and completely occupied the normal-sized pleural cavities.

The hernial sac lying in the mid-line behind the pericardium protruded through a large hiatus about four centimetres in diameter in the centre of the diaphragm. The sac contained a large liver lobe, a stomach, pancreas and spleen and a few loops of small bowel. The diaphragm was in its normal position. When the abdominal cavity was opened, a very large liver was present on the right side. Another stomach was present in the normal situation and a second pancreas was lying behind the stomach. A second spleen was present in the normal situation under the left costal margin. The small intestine was duplicated for most of its length, each gut and mesentery being joined together by loose areolar tissue. The large intestine was not duplicated and was present in the normal position in the abdominal cavity.

A normal kidney was present in each loin in the normal situation, and each was surmounted by a normal suprarenal gland (Figure IV). On the medial side, however, lying between the two vertebral columns was a suprarenal gland for each side, but no normally developed kidney. Below the suprarenal glands was a small mass of tissue

FIGURE II.

The organs of the chest. Note the fusion of the two hearts at their atria.

about one-quarter the size of the normal kidney. On microscopic examination this proved to be kidney tissue. Two fused ovaries were lying on the superior surface of this mass, between the medial suprarenal glands. The ureters were normally developed in the lateral kidneys, but a ureter could not be found leading from the central kidney mass.

The pelvis was well developed and contained the normal organs of a female fœtus (Figure IV). There was a normal uterus in the mid-line, and normal tubes and ovaries were found on each lateral aspect. The ureters from each lateral kidney were traced into the bladder, but there was no evidence of a third ureter from the medial mass of kidney tissue. The rectum was normal.

Circulatory System.

The cardio-vascular system was subjected to more detailed dissection (Figure II).

When the single pericardial cavity was opened, two hearts were seen. The ventricle of each heart was well formed, but the atria had fused in the mid-line and appeared to form one large single atrial cavity. The innominate veins on the lateral aspects entered the common atrium. The veins on the medial side of the necks fused together in the anterior part of the mediastinum and formed a large medial superior *vena cava* which passed directly into the atrium.

These veins, together with the single large inferior *vena cava*, opened into the atrium. The pulmonary veins from each set of lungs also opened into the common cavity. In the left heart the right and left atrio-ventricular valve systems were also normal. The ventricles were well developed and had a normal septum.

In the right heart there was only a single large atrio-ventricular opening. Normal valve cusps could not be seen in this orifice. The ventricle had a rather thick muscular wall, but only a rudimentary septum near the apex. The aortic orifice was on the right side and the

pulmonary artery on the left. These vessels were of normal size, and the pulmonary artery divided into its two branches to the lungs in a normal fashion. Each aorta formed its normal branches and passed round to the corresponding spinal column normally and descended to just above the region of normal bifurcation when the two trunks fused before dividing again into the common iliac arteries.

Alimentary System.

On detailed dissection of the alimentary system it was seen to be duplicated to a large extent (Figure III).

The large hernia present in the postero-medial portion of the diaphragm had caused a somewhat abnormal distribution of the organs. A single large liver was present

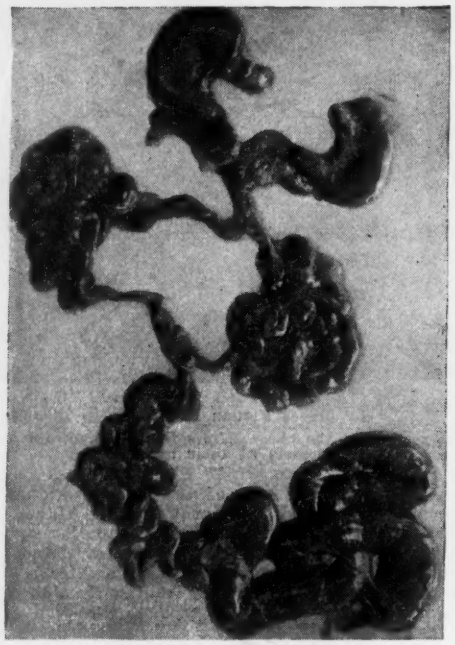

FIGURE III.
The gastro-intestinal tract.

in the normal position, but a large lobe protruded into the hernial sac, and this apparently represented part of the liver of the left half of the monster.

The stomach of the right half was also lying in the hernial sac, together with much of the corresponding pancreas of the right side. The stomach of the left fœtus was present in the normal situation beneath the single large liver, and the duodenum and pancreas were in the normal situation. The two duodenums were closely bound together by loose areolar tissue.

On the under surface of the liver were two gall-bladders separated by a short distance, and each common biliary duct was traced down to open into the corresponding duodenum. The small intestine with its mesentery was duplicated for most of its length; corresponding portions were closely bound together by loose areolar tissue. The small gut fused into a single tube a short way from the caecum. The caecum and colon and rectum were normal in shape, size and position.

Vertebral Columns.

There was a normally developed vertebral column on each side (Figure V). In the dorsal region the normal number of ribs articulated on each side. On the lateral aspect the ribs extended round to the rather widened sternum, but on the medial side they fused in a thin, central, elevated bar of cartilage lying between the vertebral columns.

In the lumbar region the vertebral columns were close together and in the pelvis the two sacra formed an arthrodesis with each other. The bony pelvis elsewhere was normally formed.

Comment.

Two years earlier a similar monster had been observed at this hospital and reported by Williams (1944). A comparison of the findings in the present and the previous case, which was available in the museum for reference, revealed a number of interesting points.

The general appearance of both monsters is, except for the characteristics of the external genitalia, practically

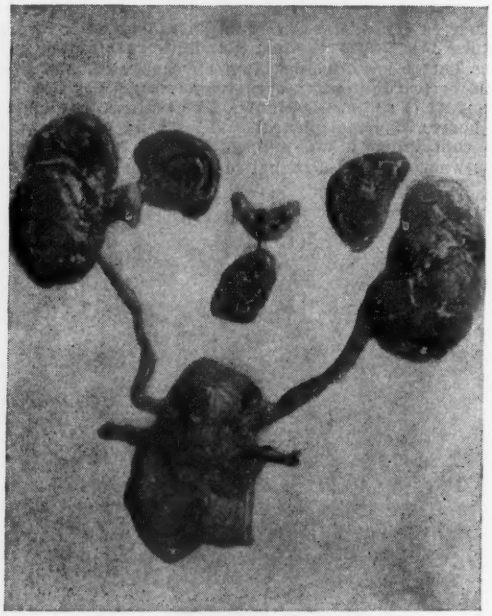

FIGURE IV.
The uro-genital system. Note in the middle of the picture the kidney remnant with the two fused ovaries above it.

identical, the only difference being the presence of a *spina bifida* of the left fœtus in Williams's case. As was therefore expected, the skeletons in both cases have the same features.

With regard to the cardio-vascular system, both monsters had a common pericardium, but while in the earlier case the two hearts were separate, in the present case the two atria were fused. There is, however, an interesting parallel: in both cases the left heart had a normal configuration, while the right proved to be extensively malformed. The respiratory system had identical features in both cases, being completely duplicated in each of them. Examination of the alimentary tract showed identical degrees of duplication in both cases; there was only some difference in the sites of the organs, due to the diaphragmatic hernia in the present case. With regard to the uro-genital system, only the lateral kidneys were developed in Williams's case; in the present monster, a remnant of the medial kidneys was found between the two vertebral columns. Although the sex of one of the subjects was male and of the other female, three gonads were found in both cases. The lateral gonads had apparently developed normally, while the medial gonads were represented by a fused non-descended organ.

Of considerable interest is a comparison of the clinical course in both cases. In Williams's case delivery of the monster was achieved only after one head and one arm had been severed from the trunk. During the manipulations the uterus had ruptured and the placenta escaped into the abdominal cavity. Although the patient was operated upon immediately after admission to this hospital, a fatal outcome could not be prevented. In the present case gentle but firm traction from below and pressure from above delivered the monster without even a perineal tear.

Cases of this kind are extremely rare. Williams's case was the first to be observed in 47,048 deliveries in this hospital. Mudaliar (1930) saw four among 25,000 deliveries at Madras. It is therefore not astonishing that a correct diagnosis is usually not made before delivery; "locked twins" are most often suspected.

FIGURE V.
The skeletal structures of the monster.

The symmetrical double monsters can be, as Sheares (1941) has pointed out, conveniently assigned to three groups: (i) twins divided at the cephalic pole only (*anterior duplicitas*); (ii) twins divided at the caudal pole only (*posterior duplicitas*); (iii) twins divided at both poles with a varying degree of fusion of the thorax, abdomen and pelvis. The present case would, of course, belong to group (i), and the common descriptive term for it is *thoracopagus tribrachius dipus*.

Summary.

A case of *thoracopagus tribrachius dipus* is described.

A brief summary of the clinical history is given.

The case is compared with a similar one reported from the same hospital some years previously.

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A CASE OF SYNCEPHALIC TWINS.

By W. SMELLIE and J. STARR,
Wynyard, Tasmania.

ON March 4, 1950, Mrs. E.A.S. gave birth to syncephalic female twins at the Spencer Maternity Hospital, Wynyard.

Mrs. E.A.S. had been examined three times before her confinement. Her last menstrual period occurred on July 4, 1949, and the last consultation was on February 24, 1950. She had suffered from severe cystitis for a month. The *fundus uteri* was at the xiphisternum. The abdominal

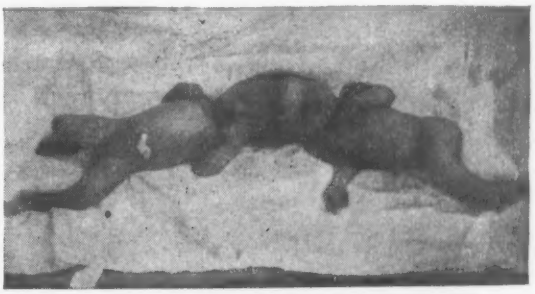

FIGURE I. Photograph of the twins.

FIGURE II. Skiagram: antero-posterior view.

wall was pendulous. Twins were not palpated. When the possibility of a twin pregnancy was mentioned, Mrs. E.A.S. said that she was no larger than in her previous pregnancies.

The patient has five children living, and had had stillborn premature twins eight years earlier. The first child is mentally defective. Mrs. E.A.S. and her husband are first cousins. The husband's sister is said to be "a pig-faced woman".

Mrs. E.A.S. was admitted to hospital at 7 p.m. in strong labour. Uterine contractions had begun at 10 a.m. Twins were diagnosed by palpation; the presenting part was a breech. There was considerable delay in the second stage, so the membranes were ruptured and a left foot was

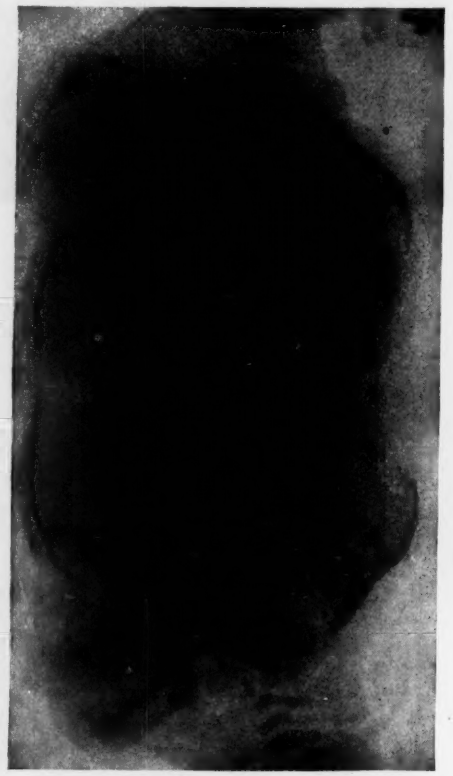

FIGURE III. Skiagram: lateral view.

brought down. Some time later the right foot presented. Delivery was still delayed. The presentation of a third foot suggested a major complication. On vaginal examination four legs were encountered. To prevent impaction one twin was pushed up beyond the pelvic brim, traction meanwhile being applied to the other. Delivery then proceeded easily. However, attached to the after-coming head was the head of the second twin; the two skulls formed one unit. The twins were very much alive.

There was one placenta and a single amniotic sac. The two cords were attached separately, but close together.

The twins weighed nine pounds eleven ounces. Their length was 33·5 inches. From the left external auditory meatus of one to the right external auditory meatus of the other the distance was 4·5 inches. With the exception of their abnormal junction together at the cranial vaults, they are normal in every respect.

Reviews.

SIGHT LIGHT AND EFFICIENCY.

LIGHTING is such an important thing in everyday life, that it is a wonder text-books on ophthalmology do not devote a chapter to the broad principles of the application of light to its many phases.[1]

The publication of such a work has apparently been left to a layman who has tried to explain the importance of lighting relative to sight and the efficiency of the work performed by people in their many and varied tasks. He has tried to give the layman an elementary knowledge of the mechanism of the eye and its functions. For this reason the book should prove useful to the lay public as well as to oculists, industrial medical officers, illuminating engineers, architects, welfare and public health officers and factory managers. It is well illustrated with pictures and diagrams.

The book has been prepared at the Institute of Ophthalmology, London, and the author has had the assistance of Sir Stewart Duke-Elder, Sir John Parsons and various lighting engineers. It is written from an essentially practical standpoint, and the writer has used the results of recent topical scientific investigations made chiefly by the Medical Research Council and the Department of Scientific and Industrial Research in England.

RHEUMATISM.

H. WARREN CROWE, senior physician of the Charterhouse Rheumatism Clinic, has published a second edition of his book entitled "Rheumatism", which first appeared in 1939.[2]

The editor has informed us in the new preface that the book has been largely rewritten because of the incorporation in the work of the clinic of the great advances made in the treatment of the rheumatic diseases in a decade which has included another world war.

Since 1912, Dr. Warren Crowe has believed that 99% of cases of rheumatism are caused by staphylococcal or streptococcal infection, and he has collected most of the evidence in support of this view, but has omitted the large amount of evidence against it. The earlier list of osteotropic bacteria contained the *Micrococcus deformans* as the sole staphylococcal representative, but two more varieties of staphylococci have been added. Under the leadership of the senior physician the "standard" vaccine therapy for chronic rheumatism is still being used extensively. He claims that it is always curative in early rheumatoid arthritis of young women and will arrest the process in 70% of osteo-arthritis. But the vaccine must be used precisely in a certain manner and, if the expected success does not materialize, the failure is due to unskilful administration. To a disciple of Crowe such terms as "response", "reaction" and "relapse" have special meanings which are incompletely understood by the uninitiated. It seems to be a great pity that this emphasis is still being placed on the use of vaccines for chronic rheumatism; there are so many other forms of therapy available which are based on better theories and sounder clinical experience. Perhaps it is true that the patients demand and expect weekly injections of some kind, and yet it is almost unethical to give them for that as a main reason. It may also be true that regular supervision and assessment of progress are in the best interest of the patient, and some such compelling reason as the giving of an injection may ensure that the patient comes to the clinic often and for a long period of months or years.

A great deal of valuable factual information is given in the book and there are some excellent sections, such as the splendid chapter by A. T. Fripp on the orthopædic treatment of rheumatism and the one on the care of the feet by O. Meyer. The use of local analgesia for non-articular rheumatism is also described satisfactorily. It is necessary to add that the clients throng the Charterhouse Clinic to the number of some three thousand visits a week; the magnitude of the experience of the staff is a guarantee that the records of that experience should be known to all practitioners who are called upon to treat rheumatic patients. There is not a section on juvenile rheumatic carditis.

[1] "Sight Light and Efficiency", by H. C. Weston; 1949. London: H. K. Lewis and Company, Limited. 9¼" × 6¼", pp. 328, with 132 illustrations, some of them coloured. Price: £2 2s.

[2] "Rheumatism", by H. Warren Crowe, D.M. (Oxon.), B.Ch., M.R.C.S., L.R.C.P.; Second Edition; 1949. London: Staples Press, Limited. New York: Staples Press, Incorporated. 8½" × 5½", pp. 270, with 26 illustrations. Price: 17s. 6d.

The Medical Journal of Australia

SATURDAY, MAY 20, 1950.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE REGISTRATION OF SPECIALISTS IN WESTERN AUSTRALIA.

THE State of Queensland undertook the registration of specialists in the practice of medicine when its Parliament passed *The Medical Act of 1939* in November of that year. The measure was described in some detail in these columns in the issue of April 13, 1940. Queensland was the first Australian State to register specialists. Various opinions are held about the advisability of specialist registration. If the standards set up for inclusion in the register of specialists are sound and if they are applied with justice and understanding there can be no great objection to the procedure. There is, however, one fact which cannot be overstated in a community which registers —in other words, brands—its specialists and it is that the possession of a higher degree or diploma in a special branch of medicine does not of itself denote the possession of special skill—the fact that the possessor of such a hall-mark has his name entered in an official book does not increase his skill. The late Frederick Treeves did a great service to medicine when he wrote his tale "The Idol with the Hands of Clay" and published it in the volume entitled: "The Elephant Man and Other Reminiscences". It is the tale of a medical man who had an F.R.C.S., an adoring young wife and no surgical experience. When she suffered from acute appendicitis he operated on her because she insisted, and the details of the tragedy are told by Treeves in a moving and unforgettable way. As she lay dying, done to death by a husband with a specialist qualification, she opened her eyes and murmured her last words: "Wonderful Boy!" This story is specially one for young doctors to read. But we must return to the subject of the registration of specialists and point out that in Western Australia specialists are now registered.

In October, 1949, the Executive Council of Western Australia authorized the issuing of an order in Council and declared certain branches of medicine and surgery to be specialties with respect to which duly qualified medical practitioners might be registered as specialists. This was done under Section 11A of the *Medical Act* 1894-1946. The branches of medicine set out in the schedule were: anæsthetics, biochemistry, dermatology, medicine, obstetrics and gynæcology, ophthalmology, orthopædics, oto-rhino-laryngology, pædiatrics, pathology, psychiatry, radiology and radiotherapy, surgery, tropical medicine, urology, and venereal diseases. A specialist is defined as "a medical practitioner who by additional study and experience in a particular branch of medicine or surgery has acquired a proficiency in that branch of a standard sufficiently high to satisfy the requirements of the Board as prescribed by Rule 25". Rule 25 is as follows:

> Any person who desires to have his name entered on the Register of Specialists shall apply to the Board in form H as prescribed in the Schedule to the rules and before he shall be registered under these rules he shall satisfy the Board—
>
> (*a*) that five years have elapsed since obtaining a qualification entitling him to registration as a medical practitioner, of which at least one year must have been passed as a resident medical officer in a general hospital or hospitals;
>
> (*b*) that he has gained and maintained special skill in that particular speciality by adequate experience in that speciality in private practice for a period of not less than five years, or in a hospital or hospitals approved by the Board for a period of not less than three years, or partly in private practice and partly in a hospital approved as aforesaid for a period of time not less than four years and has maintained such skill;
>
> (*c*) that (in the case of a person applying for registration after the expiration of one year from the date of publication of this rule in the *Government Gazette*) in addition to the above qualifications, he also possesses a higher degree or diploma in the speciality mentioned in his application, granted by a university or other institution approved by the Board provided that such degree or diploma is generally granted or recognised in such speciality. The possession of a higher degree or diploma may, in the discretion of the Board, entitle the applicant to a reduction of six months from the periods of years stipulated by paragraphs (*a*) and (*b*) of this rule.

A registered specialist may, "at the discretion of the Board", nominate at any time for a second specialty. At the same time a registered specialist will be required to confine his practice to the specialty or specialties in which he is registered. If he fails to do this, his name will be deleted from the Register of Specialists. A medical practitioner will be deemed guilty of professional misconduct if at any time after the expiration of the set period from the publication of the rule, he holds himself out to be or practises as a specialist without being registered as such. The final point is that for registration of one specialty the practitioner will be required to pay a fee of five guineas, and for registration of a second a fee of three guineas. No further fees will be payable by registered specialists in respect of their specialist registration.

The conclusion to be stated is that if specialists must be registered—we do not agree that it is necessary that they should—then the Western Australian Act is acceptable, as was the Act in Queensland. Like many other legal enactments, everything will depend on the way in which it is administered. One thing that should be appreciated is that (to quote our statement of April, 1940) the provisions are fair in that they do not close the ranks of specialism to a general practitioner; there is no clause that would limit the performance of special procedures to registered specialists. To attempt the introduction of such a provision would be wrong and stupid—to begin with, every qualified medical practitioner should be allowed to undertake any procedure of which he deems

himself capable; secondly, in a country of great distances (Western Australia is the largest Australian State) a practitioner is likely to be thrown on to his own resources and to be compelled to undertake forms of treatment which may present considerable difficulty.

THE CHARTING OF MUSCLE ACTION IN POLIOMYELITIS.

IN such a condition as poliomyelitis careful study of the patient involves both examination and estimation of the action of individual muscles. Clearly some record should be kept of the findings. This is necessary, first of all, that the identity of affected muscles should be known from the beginning of the illness, and secondly, that any change in muscular action, for better or for worse, should be recorded for future reference. It is true that a medical attendant or a physiotherapist may be able to retain a clear mental picture, correct in every detail, of a patient's condition. But doctors and physiotherapists treat many patients and it is foolish to rely on memory. Poliomyelitis, moreover, is one of those conditions which call for constant care of patients, often over many months. Patients, physiotherapists and even medical practitioners move about from place to place and patients do not always remain in the hands of the same therapists—in fact, changes in attendants may be made for all kinds of reasons. We may thus state that it is essential that careful records should be kept in every case of poliomyelitis.

There should be general agreement about a second statement, that confusion will be avoided, charting will be made easier, and thus the best interests of the patient will be served, if one type of chart is used by all medical attendants and physiotherapists. We have been informed that there are in use in the six States of the Australian Commonwealth at least four different methods of charting muscle action and four different chart forms. This has given some concern to the members of the Australian Orthopædic Association. In conjunction with certain physiotherapists, the Association has resolved to try to secure the use of a standard form throughout Australia. The chart selected as most suitable is that which is used at present in Victoria, Queensland and South Australia, with some minor alterations. For example, the size of the chart has been reduced to conform with the size of the case sheet used in most hospitals, and the order of the muscles has been rearranged. The numerical scheme for the evaluation of muscle strength, 0–6, has been retained. The form is simple and is designed to cover a period of twelve months' treatment in each individual case. The form is to be issued to hospitals in New South Wales and the Australian Orthopædic Association has expressed the hope that other States will accept the new document. This attempt to produce uniformity of action is to be commended. The Australian Orthopædic Association has members in every State; they should use their influence in the hospitals on whose staffs they serve. It would be wise to send the chart to the Branches of the British Medical Association in the several States and also to the Royal Australasian College of Surgeons and The Royal Australasian College of Physicians.

Current Comment.

CONJOINED TWINS.

IN this issue are published reports of two cases of conjoined twins. One, by W. Smellie and J. Starr (see page 667), refers to the now famous Tasmanian twins, apparently normal females in all respects except for the junction of their cranial vaults—the so-called *craniopagus parietalis*. The other, by R. J. Riddell (see page 664), concerns a female double monster with two heads and necks, a single trunk, two normal arms and a third rudimentary central arm, and a double set of thoracic organs; according to the classification recognized in "Blakiston's New Gould Medical Dictionary", this might best be described as *dicephalus dibrachius*. Both these conditions are rare, though greater interest naturally attaches to the twins which survived. How often such conditions occur is difficult to determine. B. Mortimer and J. D. Kirshbaum[1] point out that statistics on the subject are not particularly accurate. They quote a number of investigators. One found that about 7% of 100,000 pregnancies terminated in the expression of pathological ova and about 0·6% in the birth of malformed fœtuses at term; of the latter only a small portion were conjoined twins. Another investigation found 517 malformations in a series of 100,000 births investigated, but only two were double monsters. Others reported finding two double monsters in 25,000 deliveries, four in 25,000 deliveries, two in 40,000 deliveries and three in 85,000 deliveries. Thoracopagi are the most common type, and females are between two and three times as frequent as males.

Some uncertainty appears to surround the production of conjoined twins, in relation both to cause and to mechanism. It is clear that an ovum, which normally when fertilized gives rise to a single individual, may be divided and, as Mortimer and Kirshbaum state, give rise more or less to two individuals, ranging from parasitic monsters, through double monsters or conjoined twins, to two separate, truly monozygotic twins. Sir Arthur Keith[2] describes the process as taking place in relation to the centre of dominance at the anterior end of the primitive streak, the embryogenic area undergoing division either as a result of some injurious factor, a double monster resulting, or as a result of a natural or physiological process, identical twins resulting. The primitive streak, which appears on the upper surface of the embryonic plate in its hinder or caudal half about the end of the third week, indicates what is to be the mean longitudinal axis of the trunk. The anterior end of the primitive streak is the preeminent example in the embryo of an "organizer"—tissue with the power to cause certain embryonic tissues to differentiate or "organize" in a particular direction—being, in fact, the essential embryogenic area. Keith states that if the metabolic activity of this area is greatly lessened by being subjected to various noxious influences (irradiation, cold, oxygen starvation or harmful substances), then the organizing centre may be destroyed, with the result that no embryo is formed. If it is merely damaged, then two centres of dominance may appear and a double embryonic area is produced. The embryos may be fused or conjoined in many ways, giving rise to the various kinds of double monster.

Another interesting theory of origin of double monsters, originally put forward by certain Polish workers and recently referred to by R. R. Gates,[3] is that they are produced from a binucleate egg fertilized by two sperms. Gates considers that this theory probably accounts for certain types of double monsters and would explain the large differences between conjoined twins which various investigators claim to have observed. He suggests that the fact that conjoined twins are always of the same sex may be accounted for by the hypothesis that the union of

[1] *American Journal of Diseases of Children*, October, 1942.
[2] "Human Embryology and Morphology", Sixth Edition, 1948, page 65.
[3] "Human Genetics", 1946, Volume II, page 900.

the two embryos would be in the nature of a graft and that when the twin embryos were of different sex, or when they were genetically too unlike, the "graft" would not form. Investigation of the blood groups of a sufficient number of conjoined twins should, as Gates indicates, provide verification of the theory if it is correct.

Little comment can be made at this stage particularly directed to the Tasmanian twins described by Smellie and Starr. They are certainly a rarity. Though there are numerous records of conjoined twins who lived for years, even attaining adulthood, marrying and bearing children, we have been able to find no other instance of twins of the craniopagus type surviving birth. The prognosis cannot even be guessed from the data available. Whether or not the twins, normal or abnormal mentally as they may be, could find life tolerable unseparated is quite unpredictable; human nature is remarkably adaptable, especially if the "abnormal" state has always been normal for the individual. Surgical separation is a matter for mature judgement after careful investigation. Progress reports will be awaited with interest. Incidentally it is perhaps opportune here to remark on terminology. The term monster is in general use to describe practically all types of malformed fœtus. The word has a high emotional content, certainly tending to evoke disgust and perhaps fear. For a mother to learn, directly or indirectly, that she has given birth to "a monster" may cause a degree of shock and anguish little appreciated by the medical attendant who has long known the term in its conventional sense. If the fœtus is dead the harm is great enough. If conjoined twins are born and survive it is unpleasant to think that they, as well as their mother, should learn that they are monsters or, worse, "a monster". Alternative terms, such as teratism or teratosis, or the special term for the particular type, might well be used in references to a dead fœtus which might reach the mother's or father's ear; and it would be a good thing to drop the term monster altogether in relation to surviving children, in favour of terms such as conjoined twins, or the appropriate special term (as in Smellie and Starr's title), or even Siamese twins, though the latter description should perhaps be reserved for the famous Eng and Chang, Chinese who were discovered in Siam and who lived for sixty-three years. They were joined in the region of the lower part of the thorax and are variously described as a thoracopagus and a xiphopagus; they toured many countries in sideshows and are quite the best-known conjoined twins, so that even if the term Siamese twins is rightly rejected in scientific writings, it will probably persist in popular usage.

X-RAY THERAPY FOR THROMBOPHLEBITIS.

IT is interesting to find that at a time when parts of Australia are being swept by enthusiasm for dicoumarol and heparin, and for estimations of the prothrombin and fibrinogen B present in the blood, all in relation to the treatment of thrombophlebitis, in America there is a school advocating X-ray therapy for this condition. A report of their results has recently been made by C. R. Snead, J. Lasner, E. L. Jenkinson and G. de Takats,[1] who have treated 100 patients suffering from thrombophlebitis of the extremities with a short course of X-ray therapy. They describe six types of thrombophlebitis of the lower limbs: acute superficial phlebitis in preexisting varicosities, acute superficial phlebitis in veins collateral to deep venous obstruction, Buerger's disease with short segmental thrombi in upper or lower extremities, deep venous thrombosis of the leg, ilio-femoral thrombosis of the calf and subacute thrombophlebitic indurations. The first three types are fairly obvious. Deep venous thrombosis of the calf causes a tender, swollen, indurated calf, warmer than its fellow; resultant emboli are frequent. In ilio-femoral venous thrombosis ("milk leg"), it is stated, the venous obstruction is at the ilio-femoral bottleneck where the superficial and deep femoral and hypogastric veins meet; massive œdema results, explained by perivenous lymphatic obstruction or hyperplasia of the inguinal lymphatic vessels, often enlarged owing to previous infections of the foot, the groin, or the genital and pelvic organs. These are the statements in the text, but no evidence is produced in support of this explanation of "milk leg", which is usually accepted vaguely as of lymphatic origin without specific proof. Subacute thrombophlebitic indurations occur in the calf medially or posteriorly; they are due to areas of lymphostasis with round-cell infiltration, fibroblastic reaction and fat necrosis, and they may spread and encircle the lower third of the leg with a tight constricting band. The treatment given to most of the 100 patients was a short course of X-ray therapy varying between one and six treatments of 80*r* (in air). In 85 of the 100 cases the result of treatment is classified as successful, that is, the periphlebitic exudate subsided, pain was relieved and ambulation was possible. Other forms of treatment are not necessarily excluded; for example, it is stated that anticoagulants are needed in the disturbance of the clotting mechanism of the blood observed in acute thrombosis, though it is not stated how this necessity is to be recognized. Apparently a good deal is left to the coordinated judgement of radiologist and surgeon. A cautious approach to this form of therapy would seem to be indicated, but further reports will be awaited with some interest. It is difficult not to be interested in a form of treatment of thrombophlebitis when those who have used it claim that relief of pain is often dramatic and that previously bedridden patients are able to walk without discomfort.

[1] *The Journal of the American Medical Association*, December 3, 1949.

THE TOXICITY OF BERYLLIUM.

THE toxicity of beryllium has been mentioned a number of times in this journal, and more particularly in a special article on fluorescent lamps by Gordon C. Smith in the issue of August 20, 1949, and in a report of two cases of damage done by fragments of fluorescent lamps by C. M. Marshall in the issue of September 10, 1949. Both of these authors stressed the dangerous nature of broken fluorescent lamps due to the beryllium contained in the "phosphor" with which the lamps are lined. Much work has been done overseas on the harmful effects of beryllium, which with its compounds is used in a variety of trades, such as the manufacture of radio valves and of fluorescent powders as well as of fluorescent lighting tubes; it is also employed in certain industrial processes including the manufacture of alloys. It causes injury to the lung, when inhaled, to the skin and to the mucous membranes, and is a substance to be treated with respect. Those who are interested in a broad survey of its effects and a summary of the relevant literature will find these in a circular recently issued by the Defence Research Laboratories,[1] which are part of the Department of Supply and Development of the Commonwealth of Australia. The circular has been compiled by Claire R. Taylor and contains a brief introductory article, in which the main aspects of the subject are mentioned, followed by a summary of the related literature including abstracts of 63 published papers and reports. The various sections are concerned with general considerations, skin lesions, lung injury, industrial processes and hazards, methods of protection against hazards, experimental work (biological) and miscellaneous matters. Copies of the circular may be obtained free from the Defence Research Laboratories, which will also give advice on the suitability and maintenance of any particular type of dust respirator intended to protect against beryllium inhalation and on the testing of other protective equipment, as well as on related matters. Applications for copies of the circular should be addressed to the General Superintendent, Defence Research Laboratories, Private Bag Number 4, Post Office, Ascot Vale, W.2, Victoria.

[1] "The Toxicity of Beryllium", by Claire R. Taylor, B.Agr.Sc., Defence Research Laboratories, Information Circular 15; 1949. Maribyrnong, Victoria: Defence Research Laboratories. 8¼" x 5⅛", pp. 43. *Gratis.*

Abstracts from Medical Literature.

MEDICINE.

Chronic Pneumonitis with Cholesterol Deposits.

W. R. WADDELL *et alii* (*The Journal of Thoracic Surgery*, October, 1949) describe in detail ten cases of chronic non-suppurative interstitial pneumonitis in which affected parts of the lung were surgically removed and examined histologically. It was found that high concentrations of cholesterol and cholesterol esters were deposited within the pulmonary parenchyma. No tissue necrosis or bronchial stenosis could be found to account for the deposition of cholesterol, but the authors mention the possibility that during the initial pneumonia which these patients suffered, a viscid exudate caused prolonged obstruction in the bronchioles, thereby leading to their partial destruction and ectasia and chronic pneumonitis and parenchymal fibrosis of the secondary lobules beyond the obstruction.

Quinine and Influenza Prevention.

M. A. JOSSERAND (*Revue du paludisme et de médecine tropicale*, February 15, 1950) refers to the value of quinine as an influenza preventive if taken at the right time. He states that experience has proved quinine to be useless except in the very first hours of the infection, the prefebrile phase, when the subject feels that he is about to have an attack (and when in fact he has an attack). At that time influenza is suspected only because of slight pharyngeal pricking, slight headache, slight pain on pressure upon the eyeballs, and especially the impression that the cold air of the street has suddenly become hostile. The author states that a dose of quinine taken then, and if necessary repeated in a few hours, is effective in almost all cases. Taken later, it is useless.

Aureomycin.

W. W. SPINK AND E. M. YOW (*The Journal of the American Medical Association*, December 3, 1949) discuss the present status of aureomycin in the treatment of human infections. They state that staphylococci resistant to penicillin and streptomycin may respond to aureomycin. The authors report nine cases of infection with this type of organism which responded to oral aureomycin therapy. Similarly pneumococcal pneumonia and subacute bacterial endocarditis due to *Streptococcus fœtalis* or *Streptococcus viridans* may respond to aureomycin if penicillin or streptomycin fails. "Chloromycetin" was more active in typhoid fever. Bacteriæmia or urinary tract infections due to *Escherichia coli* which did not respond to other therapy sometimes responded to aureomycin. *Aerobacter aerogenes* in urinary infections may similarly respond. *Granuloma inguinale*, tularæmia and brucellosis gave better results with aureomycin than with streptomycin and sulphadiazine. Typhus of all types seems to be best controlled by aureomycin. Infectious mononucleosis and atypical pneumonia, both regarded as virus diseases, have appeared to subside under aureomycin treatment, though the number treated to date has been small. Patients with tuberculosis were not apparently benefited by aureomycin. The drug is active when given by mouth. It diffuses into the pleura, bile and cerebro-spinal fluid. An effective dose is 30 to 50 milligrammes per kilogram of body weight per day; the administration of 0·5 gramme every six hours for one to three weeks is a usual procedure. Nausea, vomiting, epigastric pain and soft fæces have been observed during treatment, and in some cases intravenous drip administration of aureomycin hydrochloride three to five milligrammes per kilogram in 250 millilitres of normal saline every eight to twelve hours for several days has been less toxic. No other toxic effects have been noted.

Cancer and Endocrine Treatment.

C. HUGGINS (*The Journal of the American Medical Association*, November 12, 1949) discusses endocrine substances in the treatment of cancer. He states that the patient with prostatic cancer benefits from five milligrammes of diethylstilbœstrol given daily. Orchidectomy is said to give better results in early carcinoma of the prostate. However, relapse occurs and the patients die in five years as a rule. There is some obscure relationship between oophorectomy and breast cancer, in that women who have been subjected to oophorectomy are less liable to breast cancer. Orchidectomy is said to cause regression in cancer of the male breast. In females testosterone propionate 25 milligrammes given daily by intramuscular injection is said to relieve symptoms for nine months or more.

Infection of Children by Tuberculous Dogs.

S. URWITZ (*Acta tuberculosea scandinavica*, Volume XXIII, Number 2, 1949) reports an instance in which five persons, including four children, who had played with a tuberculous dog developed tuberculosis. Out of 3673 dogs examined *post mortem* at the Stockholm Veterinary College, 52 were found to be tuberculous.

Cold Hæmagglutination.

J. C. BATEMAN (*Archives of Internal Medicine*, October, 1949) discusses symptoms attributable to cold agglutination. He states that agglutination of all types of red blood cells at cold temperatures by serum, with reversal at warm temperatures, occurs in a variety of conditions. In acute infections, trypanosomiasis and acute hæmolytic anæmia it is transient, but in cirrhosis of the liver, Raynaud's syndrome and some cases of acquired chronic hæmolytic anæmia, it may be permanent. Difficulty in blood counting or cross matching of blood at room temperatures, or the development of acrocyanosis and hæmoglobinuria following exposure to cold may lead to suspicion of the presence of cold agglutinins. In primary atypical pneumonia the presence of cold agglutinins has been noted frequently. Thrombosis occurs rarely. Blood containing potent cold hæmagglutinins readily hæmolyses when shaken in the cold. Normal blood does not show the same change. It had been suggested that sudden massive intravascular hæmolysis or thrombosis resulted from sulphonamide therapy, but actually such incidents occurred before these drugs were used. Cold hæmagglutinins occur in acrocyanosis, thrombotic phenomena, hæmoglobinuria and hæmolytic anæmia. Most cases occur in the third to fifth decades, and more in males than in females. It is to be noted that the methods used for determining the cold hæmagglutination titre, and for collecting and storing blood samples, are not uniform. Of 42 cases reported, there were 13 of acrocyanosis, six of thrombotic phenomena, 12 of hæmoglobinuria and 21 of various degrees of anæmia. Two cases are reported by the author, one of atypical pneumonia in which thrombophlebitis of superficial veins occurred. Cold hæmagglutination titres were 1:1280 on November 27, 1:2560 on December 2 and 1:640 on January 10. A second case was in a man, aged forty-three years, who had purple mottling of face, ears and fingers after exposure to cold in November, 1944, followed by passage of red urine. The condition recurred in cold weather. In April, 1946, his cold hæmagglutination titre was 1:2,621,440. Gross clumping was noted in oxalated blood at room temperature. The symptoms of cold hæmagglutination are due to intravascular clumping of erythrocytes. Stasis in peripheral parts of the body occurs with patchy cyanosis. These symptoms are usually due to cold. If cyanosis occurs red urine is usually passed fifteen minutes later. The redness is due to hæmoglobinuria. Salicylate therapy was tried in one of the cases reported, without benefit.

Infections of the Urinary Tract.

S. F. WILHELM *et alii* (*The Journal of the American Medical Association*, November 19, 1949) discuss infection of the urinary tract with *Aerobacter aerogenes*, the old name for which is *Bacillus lactis aerogenes*. This organism was present in 50% of patients with urinary tract infections in Beth Israel Hospital in 1948, and seven deaths resulted. It causes epididymitis and is frequently present in paraplegia when the urine becomes infected. The authors state that treatment with aureomycin 500 milligrammes given four times a day was effective in 21 out of 23 cases of urinary tract infection with this organism.

The Conjunctival Vessels in High Blood Pressure.

A. LACK *et alii* (*American Heart Journal*, November, 1949) have devised a new method of "biomicroscopy" of the conjunctival blood vessels, utilizing high magnification (up to 200 diameters). The results of a study of 100 cases of high blood pressure suggest that the capillary blood vessels are characteristically altered in this disease, the more so the higher the diastolic blood pressure. The abnormalities include extensive narrowing, elongation and looping of the capillaries, which also show fixed angularities or tortuosities, tubular thickening of the walls and loss of their normal distensibility.

Relief of Pain in the Chest by Tetraethylammonium Chloride.

H. L. ISRAEL *et alii* (*The New England Journal of Medicine*, November 10, 1949) observed that the intravenous administration of tetraethylammonium chloride to a patient acutely ill with thrombophlebitis and pulmonary infarc-

tion unexpectedly relieved the patient's severe pleuritic pain, and they were led to study the effect of this drug in other patients suffering from pain in the chest. It was found that relatively small doses (three milligrammes per kilogram of body weight) relieved pain in the chest due to a variety of conditions, including pulmonary embolism, trauma, pneumonia, pleurisy, tuberculosis, neoplasm, mediastinal emphysema and infarction of the heart. Relief was notably complete and prolonged in patients with pain due to infarction and trauma.

Atrophy of the Subcutaneous Fat following Insulin Injections.

W. S. COLLENS *et alii* (*The New England Journal of Medicine*, October 20, 1949) present a successful method of treatment of the atrophy of the subcutaneous fat which often occurs after insulin injections. A young woman under their care had developed numerous such areas of fat atrophy on the arms, thighs, abdomen and buttocks. In desperation and not knowing where else to inject her insulin she inquired whether it would be harmful to inject more insulin into the depressed, atrophic areas. No objection was raised: when she returned a month later, it was found that one atrophic area into which she had not injected insulin for two years, had after repeated daily injections filled out and returned to its normal configuration. Six other patients were advised to do the same and in every case the depressed areas were gone within a month.

Chloramphenicol and Typhoid Fever.

R. A. GOOD AND R. D. MACKENZIE (*The Lancet*, April 1, 1950) report a clinical trial of chloramphenicol in thirteen cases of typhoid fever, alternate patients being given the drug. They state that the trial showed that chloramphenicol has a specific clinical effect in this disease. Among the six treated patients, a total dosage of 19 to 22 grammes of the drug, given in eight days, did not prevent relapses or the development of the carrier state. The relapses among the treated patients occurred later than those among the untreated controls. In three cases in which chloramphenicol was given fæcal excretion of *Salmonella typhi* stopped temporarily during treatment, but started again either before or at the end of the course. No evidence was found that *Salmonella typhi* developed an increased resistance to chloramphenicol. No toxic effects of the drug were noted.

A. L. K. RANKIN AND A. S. GRIMBLE (*ibidem*) report their results in eighteen cases of typhoid fever. Nine patients were treated with chloramphenicol, nine were not so treated and may be regarded as controls, but the method of selection of patients for treatment makes the trial not a truly controlled one, as the authors had intended; they felt that it was unjustifiable to withhold the drug from the more severely affected patients with continuous pyrexia and toxæmia with prostration and/or delirium. They draw no firm conclusions, but state that the results suggest that chloramphenicol will be effective if given in adequate dosage over a minimum period of eighteen to twenty days, an initial dose of 4·0 grammes being followed by 3·0 grammes daily until the patient is apyrexial, then 1·5 grammes daily for a week, and 1·0 gramme daily for the final week. The authors consider that chloramphenicol has eased the heavy strain that typhoid fever imposes on nurses, but that the success of any treatment of this disease still largely depends on the nurses' proficiency, devotion and vigilance.

A. H. EL RAMLI (*ibidem*) analyses the results in 200 cases of treatment of typhoid fever with chloramphenicol at the Abbassia Fever Hospital, Cairo. He states that the average duration of fever after treatment started was 3·5 weeks, the relapse rate among patients followed up for at least three weeks was 27·5%, and the mortality was 6·5%. Relapses were fewest when twelve-hourly doses were given. The response to chloramphenicol was little affected by the severity of the condition or the duration of symptoms before treatment. Mild toxic effects, ascribed to the antibiotic, were observed, including anorexia and gastric upsets, stomatitis and glossitis, rash and apathy.

Aseptic Hæmogenic Meningitis.

IRA J. JACKSON (*Archives of Neurology and Psychiatry*, November, 1949) discusses the meningeal reaction to blood and its breakdown products. The work is experimental. Injections of blood or its products are made by a cisternal puncture, and the subsequent meningeal reaction is assessed by examining the cerebro-spinal fluid. Twelve main groups of substances were injected, including fresh blood, incubated blood, red cells alone, plasma, hæmoglobin and bilirubin. These experiments suggest that the agent in the blood causing the greatest meningeal response is in the hæme component and is probably bilirubin or a closely related pigment. The experiment shows that the reaction reaches a peak when the blood which has been stored for three days is injected. This coincides with the time such reactions are seen after rupture of aneurysms or after intracranial operations.

Closed Pneumonolysis.

I. A. SAROT *et alii* (*Diseases of the Chest*, November, 1949) state that Maurer's enucleation technique of closed pneumonolysis (in which the portion of the parietal pleura forming the base of the adhesion is enucleated from the chest wall) has rendered obsolete the classical method of Jacobaeus (in which the adhesion is divided). The enucleation technique has increased the scope of the operation and reduced the incidence of pleural complications.

W. W. TUTTLE (*ibidem*), discussing the paper of Sarot *et alii*, expressed the view that thoracoplasty was now often a safer and more satisfactory form of treatment than extensive intrapleural pneumonolysis.

Attempted Suicide with Protamine-Zinc Insulin.

A. VOGL AND S. H. YOUNGWORTH (*The New England Journal of Medicine*, October 20, 1949) report the case of a man, aged sixty-three years, who attempted suicide by injecting 2000 units of protamine-zinc insulin. He had few symptoms during the first twenty-four hours, but in spite of vigorous and continuous treatment there were periods of coma on the second and third days and recurrent manifestations of milder hypoglycæmia up to the sixth day. During this period he received nearly 3000 grammes of carbohydrate and yet was unable to maintain or approach a normal blood sugar level. The blood sugar level, however, was kept above critical values most of the time, and the patient suffered no demonstrable permanent damage. The authors point out that the duration of the hypoglycæmic effect of protamine-zinc insulin increases in proportion to the size of the dose.

Congenital Universal Indifference to Pain.

DAVID A. BOYD AND LOUIS W. NIE (*Archives of Neurology and Psychiatry*, April, 1949) discuss the problem of indifference to painful sensations. They describe the history of a girl, aged seven years, who throughout her life had severely mutilated herself through not experiencing any pain. She was covered in scars. She picked away the side of her nose, saying that it merely "tickled". She would put her arms on a hot stove. However, she showed emotional upset if other people were hurt or injured. Examination of the child's sensory system revealed no abnormality, and her intelligence was just below average. The authors suggest that this inability to appreciate pain can be due to two possibilities. Firstly it is akin to the aphasias, in that there is non-recognition of sensation at the highest level. Another explanation is that the highest neural structures are defective. Indifference to pain is also found when the nervous pathways or the thalamus are affected, in cortical lesions of the supramarginal gyrus on the left side, as well as in psychological conditions, as hysteria, severe emotional strain and mental defect.

Calcification of the Aorta as a Sign of Syphilis.

M. C. THORNER *et alii* (*American Heart Journal*, November, 1949) state that linear calcification of the ascending aorta in patients under sixty years of age is due to syphilitic aortitis in the large majority of cases; that is to say it is evidence of late, relatively inactive syphilis. Its presence may outweigh the diagnostic implication of a negative result from a serological test.

The Nature of Streptomycin Resistance in Tuberculosis.

A. SAËNZ AND G. CANETTI (*Revue de la tuberculose*, September-October, 1949) demonstrate that the streptomycin resistance of tubercle bacilli is a property of certain varieties of the microbe, but not of other varieties. Most tuberculous lesions contain at the beginning of treatment both the resistant varieties, which are rare, and the sensitive varieties, which are the commoner. The treatment destroys the sensitive ones, leaving the field to the resistant ones. However, lesions which are relatively inaccessible to the drug may continue to produce and discharge sensitive bacilli. The administration of a second anti-bacillary drug, such as paraminosalicylic acid or promin, along with streptomycin should, in theory, give the resistant strains a much smaller chance of survival in accessible lesions.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Queensland Branch of the British Medical Association was held at the Brisbane Hospital, Brisbane, on February 3, 1950, DR. HAROLD LOVE, the President, in the chair. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital.

Reconstruction of the Knee Joint.

DR. MILTON GEANEY showed a patient to illustrate the late results of reconstruction of a knee joint. The patient, a man, aged twenty-six years at the time of his original injury, had been run over by a truck in 1936 while riding a cycle. The rear wheel of the truck had come to rest on his right knee, pinning him down to the road. The driver, when informed of the situation, instead of releasing the brake and letting the truck roll off the patient's leg, had put it into gear, with the result that the inner surface of the patient's knee took the driving force of the truck. Examination at the time of the accident revealed that there were no soft tissues left on the medial side of the joint, the bone itself had been rasped to a depth of about a quarter of an inch, and the knee joint was full of road gravel. To save the joint seemed a hopeless proposition and Dr. Geaney decided to try an immediate plastic operation. Quite a considerable time was spent in washing out the joint until all the road gravel was removed. The frayed edges of the soft tissues were excised. In order to provide a new internal lateral ligament an incision was made from the knee upwards and towards the lateral side of the thigh, down to the deep fascia, and the superficial fascia was reflected off the deep fascia for some distance on both sides of the incision. At the top of the incision a cross cut was made in the deep fascia, measuring about four inches. A strip of this width was then reflected, until, when it was doubled down, it reached the soft deep tissues below the knee joint. It was then stitched to the surrounding deep soft tissues, thus covering the knee joint with a deep layer. Dr. Geaney said that unless that layer could be covered by skin, infection and sloughing of the layer would have followed. The problem was to cover it with skin. That was done by swinging pedicle grafts down from the skin incision which had been made for the purpose of getting the deep covering of the joint. On either side of that incision cuts were made in the skin and fat at right angles, and the pedicles of skin were swung downwards to fill in the skin gap over the inner side of the knee joint. Above those pedicles others were similarly fashioned to cover the area denuded by the first pedicles. That was repeated again. Each time the pedicles were stitched with a little increased tension, and the skin deficiency became progressively smaller. Beginning with a skin deficiency of about four inches in diameter over the knee, Dr. Geaney ended with a skin deficiency about the size of a penny at the top end of his skin incision, far removed from the knee joint. Despite the absence of chemotherapy the knee area healed by first intention with a very gratifying result. Dr. Geaney felt that the rasped raw bone on the inner side of the joint had become adherent to the fascial strip and was a factor in getting such a stable joint. He thought that he had had a great deal of luck with the case because, at the time, he possessed no experience in grafts of any kind. Dr. Geaney said that a few years later the man was timber-felling, which was no light work. When he was examined at that time, there was some lateral mobility at the joint. Dr. Geaney had not come in contact with the patient since then until he stopped him in the street two weeks before the meeting. There was now very little side play in the joint and it felt very firm. Flexion was limited to about a right angle.

Median Nerve Neuritis.

DR. LYNN WALTERS then showed a patient to illustrate a painful and not uncommon condition of the hands which responded well to treatment. The patient was a man, aged seventy-six years, who had first attended the out-patient department in March, 1949, complaining of numbness in the fingers of both hands beginning nine years previously. For six years severe neuralgic pain had been present, mainly in the thumb, index and middle fingers of both hands and noticeable especially at night. The patient's occupation for many years had been that of a fireman at a brewery, and it was only after his retirement that the symptoms first developed. Examination revealed wasting of the thenar eminence of both hands with loss of all forms of sensation in the median nerve distribution on both sides. Blebs and scars of previous burns were present between the middle and index fingers of both hands probably due to unnoticed cigarette burns. No other wasting or sensory change was noted in the arms. Dr. Walters emphasized that the point of significance in the history was the fact that the pain experienced was entirely below the wrists on both sides and in the anatomical distribution of the median nerves. Wasting was confined to the thenar eminence in the median distribution. A diagnosis was made of median nerve neuritis related to the flexor retinaculum, and on August 31, 1949, the median nerves were explored at the wrist by Dr. Stubbs Brown and Dr. D. Watson. On the right side the median nerve was found to be flattened for an inch proximal to the flexor retinaculum with a good amount of perineuritis and peritendonitis. There was thickening of the flexor retinaculum. A similar condition existed on the left side, but without flattening of the nerve. The retinaculum was divided on both sides and adhesions were freed. Dr. Walters said that within a few days the pain had disappeared, and the patient had experienced no further pain since the immediate post-operative period. There had been very little improvement in relation to the wasting, and the anæsthesia was still present.

Dr. Walters said that in 1948 a successful result had been achieved for a similar condition in a tailoress who had experienced pain for some fifteen years. Attention was drawn to the condition as it was probably not uncommon and might be confused with progressive muscular atrophy, syringomyelia or the costo-clavicular syndrome. The severe pain could be alleviated by a relatively simple operative procedure.

DR. K. HIRSCHFELD commented that the condition was similar to *meralgia paræsthetica*, in which the pain was felt on the outer side of the leg. It was interesting that four of the patients with median nerve neuritis described by Russell Brain had been tailoresses.

Carcinoma of the Submaxillary Glands.

DR. G. H. BRANDIS presented a patient to demonstrate successful block dissection of a secondary carcinomatous mass attached to the body of the mandible. The patient, a tin miner, aged sixty-four years, had been referred to the Queensland Radium Institute by Dr. D. T. R. Smith, of Stanthorpe, on November 30, 1948. He had complained of a sore on his right lower lip for two months, and a swelling on the right side of his jaw for one month. Clinical examination revealed that there was leucoplakia covering the buccal aspect of the lower lip, and a typical epithelioma on the right side of the lower lip, measuring 2·0 by 2·0 by 0·8 centimetres. There was also a large hard mass 4·5 centimetres in diameter, and almost spherical, firmly adherent to the lower aspect of the body of the mandible on the right side. No glands were palpable in the cervical chain. Dr. J. V. Duhig's report on a biopsy from the lesion on the lip was "squamous epithelioma, Grade I". X-ray investigation revealed no osseous lesion in the right mandible, and the Wassermann test gave a negative result on two occasions. Dr. Brandis said that after a consultation with Dr. A. G. S. Cooper it was decided to treat the primary carcinoma by means of a radium implant and to hold the secondary condition in check, pending subsequent surgery, by deep X-ray therapy. Radium implant was performed on November 30 by Dr. K. W. Mead at the Queensland Radium Institute. With four one-milligramme needles and two half-milligramme needles a minimum tumour dose of 5000*r* was delivered in eight and a half days. By means of deep therapy the glandular mass was given a tumour dose of 2000*r* in a period of twenty-four days. Healing of the lip occurred after the usual reaction, and the glandular mass receded slightly. Operation upon the secondary carcinomatous growth was performed on February 24, 1949, by Dr. P. Macindoe and Dr. Brandis. The right external carotid artery was ligated just above the bifurcation of the common carotid and the major portion of the right mandible was excised from a point just to the right of the mid-line to above the angle. Dr. Duhig's report on the excised mass of tissue was "squamous epithelioma invading salivary gland". Dr. Brandis said, in conclusion, that although the patient now had a cough and there was a possibility that secondary deposits had appeared in the chest, yet there had been no local recurrence in the lip, face or neck over a period of twelve months since the operation. The cosmetic and functional results were reasonably good, and the patient had been spared the pain and misery of a fungating growth. It was well worth persevering surgically in a case of that sort.

Carcinoma of Lower Part of the Œsophagus.

DR. ALAN E. LEE stated that a landmark in the advancing front against disease would be the article by Allison and Borrie in *The British Journal of Surgery* of July, 1949, on the treatment of malignant obstruction of the cardia. Those authors had shown the necessary extent of the operation for gastric cancer if it was to fulfil the requirement of removing the primary growth and its lymphatic drainage in one block of tissue. It was possible that the adoption of such an operation as that proposed would make the results of treatment of operable carcinoma of the stomach more comparable with those of carcinoma of the rectum than they were at the present time. Using as illustration a patient, aged sixty-one years, in whom a carcinoma of the lower part of the œsophagus had been resected three weeks earlier, Dr. Lee pointed out, firstly, the virtues of the abdomino-thoracic incision in obtaining adequate access to the stomach and lower part of the œsophagus. While for almost all growths of the body of the stomach total gastrectomy was necessary, he believed that it was better for the patient if that could be achieved with only an abdominal incision rather than with a combined opening of the abdomen and thorax. His experience with œsophago-jejunostomy had shown that end-to-end anastomosis by the method of Yudin was compatible with a smooth convalescence, and seemed less likely to be associated with necrosis than was operation involving the wall of the stomach. Because of the liability of junctions with the œsophagus to leak, a rubber tube was always placed with its end close to the anastomosis, and in two recent patients, including the one shown, such a leak had occurred, but was rapidly controlled by placing increasing resistance on the drainage tube. Dr. Lee concluded by stating that the proper access gained by the combined incision was rapidly teaching surgeons that they must make their gastric resections for cancer more radical than in the past, and that total removal of the stomach was compatible with a quite tolerable existence, which, if not normal, was infinitely preferable to the state from which the patient had been rescued.

(To be continued.)

NOTICE.

THE Special Group on Aviation Medicine will hold a combined scientific meeting and annual general meeting on June 2, 1950, at Brisbane, during the Seventh Session of the Australasian Medical Congress (British Medical Association). Visitors to congress who wish to join the group are invited to attend.

Correspondence.

PELMATOSCOPY: A METHOD OF IDENTIFYING THE NEWLY BORN.

SIR: The above was the subject of an editorial which appeared in an issue of THE MEDICAL JOURNAL OF AUSTRALIA, dated April 15, 1950, page 509.

As the originator of a similar system of identification of the newly born in operation at the Royal Hobart Hospital, Tasmania, I would welcome an opportunity to bring to the notice of subscribers to THE MEDICAL JOURNAL OF AUSTRALIA reasons why my faith in such a system is well founded. It is obvious that the author of the editorial was not fully informed of the extent to which this system is operating in Tasmania.

For more than half a century in all parts of the civilized world, the science "dactyloscopy" has been recognized as a reliable and unforgeable means of personal identification, and is accepted by the highest authorities of jurisprudence. Identification of the individual by palmprints is frequently submitted in courts of law, and readily accepted. We have now arrived at the most important question: "Are prints of the soles of the feet as reliable as fingerprints and palmprints for personal identification?"

To answer this question we must first decide if the skin on the palms, fingers and soles of the feet is the same organ. All who have made a study of the skin and its functions will agree that it is the same organ.

The papillary ridges appear with equal prominence on the palms of the hand and soles of the feet. In both surfaces will be found all nine (9) pattern types found in the finger pulps, such as loops, whorls *et cetera*, also the ridge characteristics on which fingerprint experts rely for identification. It is generally accepted that fingerprint authorities have little or no cause to concern themselves with footprint identification, and those without experience in this field could not be expected to do otherwise than reserve judgement on the reliability of identification by this means.

Over the past nine (9) years I have gained considerable experience in examining and identifying prints from the soles of the feet in many age groups, including the newly born. I have arrived at the conclusion that the only change which takes place in the ridge formations is that they strengthen by natural growth and usage, and that the ridge characteristics remain unchanged. I am of the opinion that for identification purposes footprints are of equal value as are fingerprints and palmprints.

Experience has proved that identifiable footprints can be obtained from the newly born immediately after delivery. This is being done consistently at the Royal Hobart Hospital by members of the staff after a short period of training in the correct method. As proof of this Dr. J. C. Laver, Superintendent of the Royal Hobart Hospital, and Sister F. I. Taylor, in charge of the maternity section, selected at random a number of forms on which mothers' fingerprints and babies' footprints had been recorded several days previously. The fingerprints of the same mothers were recorded on blank forms, and footprints of their babies were purposely mixed on these forms. A careful record was compiled of the way the changes had been made, and the key to the mix-up was held by the hospital authorities.

Dr. Laver delivered the originals to me, also the forms on which the changes had been arranged, and requested that the babies be restored to their rightful mothers by footprint identification. This was the first extensive test of this system requested by the hospital authorities.

The identifications were made at the Fingerprint Branch of the Police Department, Hobart, and the forms were returned to the hospital authorities. In my presence the key to the mix-up was checked against the identifications, and it was declared that each baby had been correctly identified and restored to the rightful mother by the identification of fingerprints and footprints only.

I claim this is the answer to the question of identification of the newly born referred to in the editorial.

Yours, etc.,

Officer-in-Charge. T. G. A. STEWART.
Fingerprint Bureau, Police Department,
Hobart.
Undated.

PREGNANCY TESTS.

SIR: Regarding Dr. Bettinger's article dealing with pregnancy tests in the journal of April 15, I cannot agree that false positive results are frequently unexplained and must express disagreement with his difficulty in reconciling a false positive result with tubo-ovarian abscess.

It has been known for many years that secretions of many glands have the property of inhibiting the specific "-tropic" secretions of the anterior lobe of the pituitary acting on the particular endocrine gland. Much stress has been laid on this endocrine inhibition, particularly with regard to the thyreoid and gonads. The best example of this is the case in which orchidectomy is performed for carcinoma of the prostate; the inhibitory influence of the male testicular hormone on the anterior lobe of the pituitary is removed, and this leads to a steadily progressive increase in output of pituitary gonadotropic hormone which, with the passage of time, stimulates the suprarenal cortex to activity, and its output of male sex hormones becomes sufficient to reactivate the prostatic carcinoma.

Similarly the ovarian secretions are active as a check on the gonadotropic output of the anterior pituitary lobe. Pregnancy tests depend on the presence of large quantities of gonadotropic hormone in the urine. This may be either pituitary or chorionic gonadotropin. Excess quantities of chorionic gonadotropin are present in pregnancy and chorionepithelioma. Ovariectomy, destructive lesions of the ovary and particularly the menopause are conditions in which the inhibitory activity of the ovarian secretion on the pituitary is lacking with subsequent excess production of pituitary gonadotropin. These conditions which may give rise to "false positives" may be distinguished from pregnancy by determining whether the excess gonadotropin is of pituitary or chorionic origin. The only test at present known to make this distinction is that in which ova are produced by the female Xenopus toad in response to the injection of

chorionic gonadotropin. This means that, excluding the female Xenopus test, "false positives" may be expected in female castrates.

The most important pitfall in the use of biological tests for pregnancy occurs in the case of the menopausal woman who, suspecting she is pregnant, has an Aschheim-Zondek or male Xenopus test performed with a positive result due probably to her excess pituitary gonadotropin production.

Yours, etc.,

PAUL B. HAGEN.

Department of Physiology,
University of Sydney,
Sydney.

April 24, 1950.

SIR: I have read with interest the review of pregnancy tests by Dr. Hans Bettinger in THE MEDICAL JOURNAL OF AUSTRALIA for April 15, and would particularly commend his comments on false negative results due to failure to observe proper precautions to ensure an adequate concentration of hormone in the urine. In view of the relative frequency of false positive reactions, it is our practice to repeat the test a week later, free of charge, if the practitioner considers that the result is contrary to clinical indications.

I am surprised, however, by Dr. Bettinger's comments on the Xenopus test, which has been used in this laboratory for the past three years, and has proved very satisfactory. Experience has shown us that it is not necessary to keep the water in the tanks at a constant temperature under the climatic conditions existing in Adelaide. The equipment is simple, and consists of two large storage tanks, about twenty small tanks holding up to a dozen frogs each, and glass jars in which the tests are performed. The care of the frogs does not involve any more time or labour than that necessary for any other laboratory animal. Up to the present we have had no abnormal losses of frogs other than in association with tests, and in the majority of these cases it has been when an extract of heavily contaminated urine sent from the country has been injected.

There is no difficulty in obtaining supplies of *Xenopus lævis*, which we import through Stokes and Company, of Melbourne, who have been most helpful in maintaining our stocks. The frogs are received in special containers holding about 100 frogs, and only on one occasion was there a loss *en route* due to improper feeding. Up to the present we have received 975 frogs through Stokes and Company, of which 475 are still in use. Each frog gives an average of five tests, and as they cost about 7s. 6d. per head, inclusive of landing costs, their use is far more economical than that of laboratory bred rabbits. Recently Mr. Hansen, senior technician in the division concerned, successfully raised a small batch of tadpoles to the frog stage, and next spring he hopes to carry out some more extensive breeding experiments.

I record these facts to correct the impression that the Xenopus test is impracticable in Australia, and not to suggest that the use of male frogs may not have advantages, especially in hospitals which do not have a large animal house and staff, such as is available at this institute.

Yours, etc.,

ORDE POYNTON.

Institute of Medical and Veterinary Science,
Adelaide,

April 18, 1950.

TREATMENT OF LUNG ABSCESS.

SIR: In the journal of April 22 there appears a reference to the treatment of acute lung abscess in the "Current Comment" section. Certain statements in this I feel call for comment.

This article advocates more use, and even implies the exclusive use, of "conservative procedures" in the treatment of acute lung abscess. The word conservative has the comforting sound of essential rightness, but when conservatism is carried to the extent of becoming a cult, I submit that the use of the word is to be deprecated.

An acute lung abscess is not a specific entity because a multitude of causes, and infective organisms, play a part in its pathology. Indeed the condition may be of secondary importance to some other condition, such as a bronchial carcinoma, where the occurrence of an abscess, with fever and toxæmia, is an indication, rather than a contra-indication, for operation. In contrast to this, an acute staphylococcal abscess in the lung should never be treated by operation because if the initial period of toxæmia is overcome with the aid of the antibiotics, the condition will resolve.

The aims of treatment of an acute lung abscess follow general surgical principles, that is, to promote resolution, or failing this, to promote localization. The response of the patient to treatment, the nature and virulence of the infecting organisms, as well as the underlying cause, are factors which may decide that it is wise, or even life-saving, to operate. When localization has occurred, drainage must be effected either by natural or by surgical means, or the diseased area may be resected. The antibiotic drugs in no way alter this fundamental principle. An abscess which contains a large lung slough presents the same problem as an area of osteomyelitis which contains a sequestrum, and in both cases the indication for operation is absolute. It is true that both types of sequestra may be dealt with by Nature, the one being liquefied and coughed up over the course of months, the other being extruded through a sinus after years, but both processes entail much suffering and illness, as well as carrying definite risks to life.

Every case of acute lung abscess should be reviewed carefully every two or three days, and if the patient is responding badly to treatment, if toxæmia is becoming overwhelming, or if some absolute indication for operation can be demonstrated, such as a large lung slough, so-called "medical" treatment should be abandoned forthwith. Under such circumstances to wait for some arbitrary period to elapse is absurd.

In the article under consideration the name of the London Chest Hospital is given, and it is stated that there "a medical regimen has come into sole use".

The report is based on a study of a small series of 32 cases, of which 25 were selected for non-surgical treatment. The most favourable interpretation which can be put on these figures is that 15 out of 19 cases treated by high dosages of penicillin (2,000,000 units daily) were cured. The fate of the remaining 20% is not stated. One can only observe that any line of treatment which fails in one-fifth of the cases cannot be regarded with complacency. The clinical material of a special hospital is liable to give a false picture of any given condition because selection has occurred before patients are admitted to such institutions. At the Brompton Hospital, London, for example, an acute lung abscess, except those caused by bronchial carcinoma, is a comparative rarity, whereas a subacute or chronic lung abscess is comparatively common. Working on the surgical side, the failures of non-surgical treatment are likely to be over-emphasized, but this is because most cases are successfully treated in general hospitals.

A warning should be sounded about the use of physiotherapy in the acute stage of a lung abscess. Although its importance cannot be over-emphasized in the treatment of inflammatory processes in the chest, it should not be used when the patient is gravely ill and coughing large quantities of pus, because of the risk of drowning the sound lung.

With regard to surgical drainage, the indications for its use are becoming less and less. If used, it should be done early, and as a life-saving measure—not when the case is reaching the chronic stage. Resection is now used more and more and is certainly the only logical treatment for a chronic lung abscess.

As a final analogy it is worthy of consideration that if all cases of acute appendicitis were treated by "medical" means, the mortality would probably hardly exceed 10%, but timely operation has reduced the mortality to about 1%. With regard to acute lung abscess, one might say to the surgeon that most cases, even before the days of chemotherapy, got better, and to the physician one might say that he should consult his surgical colleague more often if the results of treatment are to be improved.

Yours, etc.,

IAN MONK.

The Royal North Shore Hospital of Sydney,
Crow's Nest,
New South Wales.

May 3, 1950.

OSTEITIS DEFORMANS IN A BROTHER AND SISTER.

SIR: Dr. Warren Murphy's report of *osteitis deformans* in a brother and sister (April 15, 1950) touches on an important aspect of a fascinating disease. Much has been written (Koller, 1946; Dickson, 1945) on the familial incidence of Paget's disease, which has been reported in identical twins (Dickson, 1945). The hereditary behaviour resembles that of a recessive gene.

I have been following a male patient with Paget's sarcoma, whose sister has *osteitis deformans*, and by a strange coincidence a sister-in-law is also affected. This latter female has no offspring. The father of the brother and sister had Paget's disease.

Yours, etc.,

MAX LAKE,
Surgical Registrar.

Brisbane Hospital,
Brisbane,
April 20, 1950.

References.

Koller, F. (1946), *"Über die Heredität der Ostitis deformans Paget", Helvetica medica acta*, Volume XIII, page 389.

Dickson, D. D., Camp, J. D., and Ghormley, R. K. (1945), "Osteitis Deformans. Paget's Disease of Bone", *Radiology*, Volume XLIV, page 449.

TREATMENT OF VOMITING OF PREGNANCY BY ŒSTROGENINE COMPOUND.

SIR: I was interested to read Dr. Whyte's report on treatment of vomiting of pregnancy by œstrogenine compound. Over a period I have used Parke, Davis "Lipolutin"—one cubic centimetre, one milligramme (one international unit per cubic centimetre) by intramuscular injection at weekly intervals. I think that a conservative estimate would show that 150 patients with nausea or vomiting have been given the injections, and of them not more than about 15 have required more than three injections. Usually the "Lipolutin" has been given in association with half a grain of "Gardenal" two or three times a day. Both methods may be more fully tried in practices with greater numbers of ante-natal cases. For myself, my results have been gratifying, but I hope they may be more so by using "O.C.P." in addition to "Lipolutin".

Yours, etc.,

DONALD F. FARMER.

Russell Street,
Toowoomba,
Queensland.
April 5, 1950.

Obituary.

GUY PERCIVAL UNDERDOWN PRIOR.

WE are indebted to Dr. Oliver Latham for the following appreciation of the late Dr. Guy Percival Underdown Prior.

Guy Percival Underdown Prior arrived in Adelaide from London in 1899. He married and was for a time engaged in hospital practice, but then took an appointment at a naval hospital in the hills of Fiji. His health having given way, he returned to Australia, and after a short time at Penola in South Australia, he formed the mental hospital service at Callan Park, New South Wales, in 1902. In time he assumed the superintendentship of Rydalmere and Parramatta Mental Hospitals. At one stage he was acting Inspector-General of Mental Hospitals. At Rydalmere, working with the late Evan Jones, he carried out many biochemical researches on the young epileptic patients in their charge: he and Evan Jones achieved quite a reputation by reason of their treatment. They had to estimate the serum calcium content with a hæmocytometer, but procured a good colorimeter for determining the amount of the other ingredients. They also studied endocrine disorders, and a packed meeting at the British Medical Association rooms roundly applauded an exhibition of patients of various endocrine types which they presented at a lecture. But his crowning work was initiated at Parramatta—the artificial fever treatment of mental and physical diseases, such as general paralysis of the insane, lightning pains of tabes and syphilis itself in conjunction with other remedies, the result being checked by the findings both in the serum and in the cerebro-spinal fluid, as well as by the clinical condition. Prior had long been in communication with Professor Clarence Neymann, of North-West University, Chicago, who was the originator of the heat treatment and claimed to have relieved the lightning pains of 97% of tabetics. Prior installed the necessary apparatus for the treatment in Parramatta Mental Hospital, the first in Australia, and had considerable success with it for tabes and general paralysis of the insane and, as an adjuvant, for syphilis in various stages. In due course Evan Jones introduced it into Broughton Hall, where it was constantly in use. Professor Neymann visited Australia, with his wife, a few years ago, and the psychiatrists of Sydney were delighted to hear him recount at a dinner his early experiences with the treatment and his great pleasure in meeting Prior, who had so successfully introduced his methods to Australasia.

Prior was a born friend and confidant of mental patients and their friends, and his memory was surprising. With his pony and dogs he not only knew every inch of his grounds, but travelled far to keep contact with patients long cured and discharged. As a form of rehabilitation work, with Dr. Kinna, he transformed an old rubbish heap beneath a quarry into beautiful gardens enclosing a hockey ground and several tennis courts, and the artistic stone walls enclosing them make a magnificent approach to Parramatta Mental Hospital. He loved trees and planted thousands of flowering types both inside and outside his institutions.

He is survived by a widow, a son, who is pathologist to the County of Warwick, England, a daughter, who is the wife of Dr. A. J. M. Purchas, of Young, and six grandchildren, one an arts graduate of the University of Sydney and two medical students.

BRONTE CREAGH SMEATON.

WE are indebted to Dr. J. M. Bonnin for the following appreciation of the late Dr. Bronte Creagh Smeaton.

Bronte Creagh Smeaton was born on November 15, 1905, at Mount Barker, South Australia, the elder son of Dr. Bronte Smeaton. He was educated at Queen's College, and graduated M.B., B.S. at the University of Adelaide in 1928. After completing a year's residence at the Royal Adelaide Hospital, he went to the United Kingdom where he obtained the diploma of F.R.C.S. (Edin.). On his return to Adelaide he worked in general practice at Dulwich and later at St. Peters, at the same time gaining further experience in surgery from his appointment as clinical assistant to the surgical section of the Royal Adelaide Hospital. In 1932 he married Morna Dobbie. Just prior to the outbreak of war he met with an accident, which resulted in the loss of an eye. This most unfortunate occurrence necessitated the termination of a promising surgical career. It also turned his attention to a further specialty, radiology, a sphere in which he quickly achieved fame in South Australia. In 1940 he became the third member of the team of J. S. Verco, C. Gurner and B. C. Smeaton, a very happy association in which he practised his specialty until his death. His honorary appointments were honorary assistant radiologist to the Royal Adelaide Hospital, honorary assistant radiologist to the Adelaide Children's Hospital, and clinical assistant to the radio-therapeutic department of Royal Adelaide Hospital. He was precluded from war service by injuries sustained earlier in his life, but during the war years he worked unceasingly in his honorary capacity at the teaching hospitals, and bore the brunt of the work of the partnership practice, his partners being engaged in war service. This sustained effort contributed towards his untimely end.

Creagh Smeaton was one of the most universally liked and respected medical men of his generation. He endeared himself to his colleagues and his patients alike, a quality engendered by his pleasant personality, his ready wit, his continually buoyant spirit and his healthy, optimistic outlook on life. In his specialty his standard of work was high, and his background of general practice and surgery stood him in good stead when, having given an opinion on a radiological problem, he was asked for his opinion on treatment. Such an opinion was invariably characterized by sound practical reasoning. In the latter years of his life he carried out his duties to the full, in spite of an increasing physical disability, about the outcome of which he had no false illusions. His sudden death on March 19, 1950, at the age of forty-four years, was a serious loss to the medical profession in South Australia, and the loss of a personal friend to all of the many medical men with whom he had worked.

To his parents, Dr. and Mrs. Bronte Smeaton, and to his widow and three children, we wish to extend our deepest sympathy.

Two senior physicians have written as follows: "I was a great admirer and friend of Creagh's and my world will be the sadder with his going. He was always so cheerful and game in spite of his handicap and the certain knowledge that he could not expect to live a normal span of life. He will be a great loss to the profession—both from an academic point of view, but even more so from what I can only term the ethical point of view—as he had all the right

ideas and carried out what I fear is a fast dying standard of professional conduct."

"Everyone realizes that he had a number of misfortunes to put up with, and also how he made light of them, and adapted his life successfully to his disabilities. He will be a great loss to the medical community, as well as his circle of friends, as he was one of the few forceful downright personalities who are concerned with the truth rather than personal gain."

GUSTAVE HEUZE HOGG.

WE regret to announce the death of Dr. Gustave Heuze Hogg, which occurred on May 7, 1950, at Launceston, Tasmania.

WILLIAM HENRY FITCHETT.

WE regret to announce the death of Dr. William Henry Fitchett, which occurred on April 21, 1950, at Elsternwick, Victoria.

PERCIVAL THOMAS SPOWER CHERRY.

WE regret to announce the death of Dr. Percival Thomas Spower Cherry, which occurred on April 28, 1950, at Alberton, South Australia.

The Royal Australasian College of Physicians.

WUNDERLY TRAVELLING SCHOLARSHIPS.

THE Royal Australasian College of Physicians proposes to award two Wunderly Travelling Scholarships in 1951 for the post-graduate education and training abroad of young physicians in the diagnosis and management of thoracic disease. The value of each scholarship is one thousand pounds Australian currency. The scholarship will be tenable by graduates in medicine of a university approved by the College, who hold a Doctorate of Medicine and/or a Fellowship or Membership of The Royal Australasian College of Physicians or of the Royal College of Physicians of London, but the holding of a higher degree or diploma is not an absolute condition of the award.

Information relating to the scholarships may be obtained upon application to the Honorary Secretary of the College at 145 Macquarie Street, Sydney.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 23, of April 27, 1950.

AUSTRALIAN MILITARY FORCES.

Royal Australian Army Medical Corps.

To be Captain, 14th February, 1950.—VFX700110 Dorothy Virginia Murphy.

Citizen Military Forces.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical): To be Lieutenant-Colonel (provisionally), 1st May, 1950.—2/126984 Captain (Temporary Lieutenant-Colonel) R. E. Wherrett.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical): To be Lieutenant-Colonel (provisionally) and retain the temporary rank of Colonel, 1st May, 1950.—3/50055 Major (Temporary Colonel) R. Officer.

Royal Australian Army Medical Corps (Medical).—3/101804 Major T. P. Crankshaw is transferred to the Reserve of Officers (Royal Army Medical Corps (Medical)) (3rd Military District), 20th January, 1950.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—4/31964 Captain G. B. Fisk is appointed from the Reserve of Officers, 17th February, 1950.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

Brian Capper, B.D.S. (267878), is appointed to a short service commission, on probation for a period of twelve months, 6th February, 1950, with the rank of Flight Lieutenant.

The following officers are appointed from the Reserve to short service commissions, on probation for a period of twelve months, 6th February, 1950, with the rank of Flight Lieutenant: Flying Officer (Temporary Flight Lieutenant) E. C. Hey, D.F.C., B.D.S. (412592), Flying Officer J. F. Howell-Price, B.D.S. (411914).

The following are appointed to short service commissions, on probation for a period of twelve months, 6th February, 1950, with the rank of Flight Lieutenant: Bryan Norman Turner, B.D.S. (444663), Robert George Sharp, B.D.S. (443946).

Congresses.

FIRST INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

THE First International Congress on Diseases of the Chest will be held in Rome, Italy, from September 17 to 20, 1950. It is sponsored by the Council on International Affairs of the American College of Chest Physicians and the Carlo Forlanini Institute of Rome.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners.

Hay, Judith Alison, M.B., B.S., 1950 (Univ. Sydney), 32 Concord Road, Strathfield.

Hennessy, William Bertram, M.B., B.S., 1950 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Hensley, William Joseph, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Hobbs, Colin Arthur, M.B., B.S., 1950 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Holmes, Alan Boyd, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Howell, David, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Hunter, Irvine John, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Ireland, Basil John, M.B., B.S., 1950 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Jacks, Laurence Maurice, M.B., B.S., 1950 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Keating, Bruce Ivor, M.B., B.S., 1950 (Univ. Sydney), Lewisham Hospital, Lewisham.

Kelly, Douglas Heron, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Kyneur, Frederick James, M.B., B.S., 1950 (Univ. Sydney), Dulce Domum, Ross Street, Toongabbie West.

Lancaster, Norman Edgar, M.B., B.S., 1950 (Univ. Sydney), Marrickville District Hospital, Marrickville.

Lance, James Waldo, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Lappin, Frank, M.B., B.S., 1950 (Univ. Sydney), Parramatta District Hospital, Parramatta.

Larum, Cornelius Lloyd, M.B., B.S., 1950 (Univ. Sydney), Mater Misericordiæ Hospital, Crow's Nest.

Law, David John, M.B., B.S., 1950 (Univ. Sydney), Canterbury District Memorial Hospital, Campsie.

Leckie, Bruce Douglas, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Leech, Mabel Catherine Anne, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Leitch, David Stanley, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Lindsell, Charles Kenneth, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Line, Daphne Harrington, M.B., B.S., 1950 (Univ. Sydney), Rachel Forster Hospital for Women and Children, Redfern.
Lines, Verlie, M.B., B.S., 1950 (Univ. Sydney), Balmain and District Hospital, Balmain.
Lisyak, John, M.B., B.S., 1950 (Univ. Sydney), Parramatta District Hospital, Parramatta.
Loneragan, Thomas Paul, M.B., B.S., 1950 (Univ. Sydney), Lewisham Hospital, Lewisham.
McBride, William Griffith, M.B., B.S., 1950 (Univ. Sydney), St. George Hospital, Kogarah.
MacDonald, Colin Bruce, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
McDonald, George Roy William, M.B., B.S., 1950 (Univ. Sydney), Royal South Sydney Hospital, Zetland.
McKenzie, Nancy, M.B., B.S., 1950 (Univ. Sydney), Bathurst District Hospital, Bathurst.
McRae, Margaret Ruth, M.B., B.S., 1950 (Univ. Sydney), 31 Vaucluse Road, Vaucluse.
Markus, John George, M.B., B.S., 1950 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Marnie, Philip Muir, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Martin, Gwenda Elizabeth, M.B., B.S., 1950 (Univ. Sydney), Sydney Hospital, Sydney.
Maybloom, Bernard Laurence, M.B., B.S., 1950 (Univ. Sydney), Sydney Hospital, Sydney.
Mellick, Richard Anthony, M.B., B.S., 1950 (Univ. Sydney), Lewisham Hospital, Lewisham.
Miles, Derek Frazer, M.B., B.S., 1950 (Univ. Sydney), Gumbooya, Old Pittwater Road, Brookvale.
Mitchell, Colin Ashton, M.B., B.S., 1950 (Univ. Sydney), Saint Vincent's Hospital Darlinghurst.
Mitchell, Shirley Annette, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Morris, Marion Erica, M.B., B.S., 1950 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Nelson, Norma Majorie, M.B., B.S., 1950 (Univ. Sydney), Eastern Suburbs Hospital, Bondi Junction.
Ohlsson, Keithley, M.B., B.S., 1950 (Univ. Sydney), 10 Carnarvon Road, Roseville.
O'Sullivan, Eibhlin Maire Eilis, M.B., B.S., 1950 (Univ. Sydney), 13 Black Street, Vaucluse.
Owen, Francis Septimus, M.B., 1950 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Parkin, Gordon Maurice, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Pearson, Bruce Samuel, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Phipps, Walter James Flower, M.B., B.S., 1950 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Pittorino, Anthony Joseph, M.B., B.S., 1950 (Univ. Sydney), 141 Forest Road, Arncliffe.
Pollard, Brian James, M.B., B.S., 1950 (Univ. Sydney), Sydney Hospital, Sydney.
Powrie, Neville John Desmond, M.B., B.S., 1950 (Univ. Sydney), 15 Bradley's Head Road, Mosman.
Reimer, John Edward, M.B., B.S., 1950 (Univ. Sydney), Sydney Hospital, Sydney.
Richards, John Grahame, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Roach, Trevor Hunter, M.B., B.S., 1950 (Univ. Sydney), Canterbury District Memorial Hospital, Campsie.
Roper, John Ramsay, M.B., B.S., 1950 (Univ. Sydney), St. George Hospital, Kogarah.
Rosengarten, Lionel, M.B., B.S., 1950 (Univ. Sydney), Sydney Hospital, Sydney.
Ross, Judith May, M.B., B.S., 1950 (Univ. Sydney), Sydney Hospital, Sydney.
Sander, Ralph, M.B., B.S., 1950 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Saunders, Richard John, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 29, 1950.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	..	..	..	..	..	..	..	..
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	1(1)	1(1)	1	..	1	..	..	..	4
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	4(4)	..	..	..	..	..	4
Diphtheria	11(7)	30(7)	6(2)	4(4)	1	..	..	..	52
Dysentery (Amœbic)	*	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	*	4(3)	5(1)	..	..	..	..	..	9
Encephalitis Lethargica	..	..	..	..	1(1)	..	..	..	1
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	1(1)	*	*	..	*	*	..	1
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	..	..	..	..	..	..	..	..
Measles	*	*	*	10(8)	*	*	..	4	14
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	20(16)	3	1(1)	30(26)	2	*	*	..	56
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	2(2)	..	..	..	..	2
Rubella(*c*)	*	*	..	*	1(1)	*	..	..	1
Scarlet Fever	28(20)	24(14)	5	5(1)	5(4)	7	..	1	75
Smallpox	*	..	..	..	..	..	..	..	..
Tetanus	*	..	1	*	*	*	*	*	1
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	15(10)	4(4)	2(2)	8(7)	6(4)	1	..	1	37
Typhoid Fever(*e*)	1(1)	..	..	1(1)	..	..	..	..	2
Typhus (Endemic)(*f*)	1(1)	..	2	..	2(2)	..	..	..	5
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	*	*	*	..	*	*	*	*	..
Whooping Cough	*	*	*	2(2)	*	*	..	1	3
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.
(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Schneeweiss, Joachim, M.B., B.S., 1950 (Univ. Sydney), Marrickville District Hospital, Marrickville.
Skalla, John, M.B., B.S., 1950 (Univ. Sydney), Sydney Hospital, Sydney.
Summerbell, Edward Britton, M.B., B.S., 1950 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Tarn, Ruth Radcliffe, M.B., B.S., 1950 (Univ. Sydney), 4 Clifford Street, Gordon.
Thom, Alistair Sorley Cumming, M.B., B.S., 1950 (Univ. Sydney), Marrickville District Hospital, Marrickville.
Thomas, Barrie Adrian, M.B., B.S., 1950 (Univ. Sydney), Western Suburbs Hospital, Croydon.
Thompson, Janet Fraser, M.B., B.S., 1950 (Univ. Sydney), 17 Norwood Avenue, Lindfield.
Thomson, Roma Clare, M.B., B.S., 1950 (Univ. Sydney), 70 Bradley's Head Road, Mosman.
Thwaites, Cedric Francis, M.B., B.S., 1950 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Toakley, Natalie, M.B., B.S., 1950 (Univ. Sydney), Eastern Suburbs Hospital, Bondi Junction.
Tofler, Oswald Boaz, M.B., B.S., 1950 (Univ. Sydney), Sydney Hospital, Sydney.
Walker, Thomas Bridson, M.B., B.S., 1950 (Univ. Sydney), Auburn District Hospital, Auburn.
Watson, Lyal Clifton Albert, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Way, Stephanie Susie Jay, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Westphalen, John Brock, M.B., B.S., 1950 (Univ. Sydney), 8 Gainsborough, Marion Avenue, Rose Bay.
Wilkinson, Thomas, M.B., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Wolfers, David, M.B., B.S., 1950 (Univ. Sydney), 3 Bellevue Park Road, Bellevue Hill.
Woods, William Cleaver, M.B., 1950 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Brown, Donald Montague, M.B., B.S., 1924 (Univ. Melbourne), F.R.A.C.S., 1932, Echuca, Victoria.
Jarman, Ronald, M.R.C.S. (England), L.R.C.P. (London), 1926, D.A., 1935, F.F.A. (R.C.S.), 1949, c.o. Dr. J. S. MacMahon, 143 Macquarie Street, Sydney.
Severn, Carmen Myburgh Millott, L.M.S.S.A., 1946 (London), 5 Captain Piper's Road, Vaucluse.
Thomson, David Scott, M.B., B.S., 1929 (Univ. Melbourne), Pambula.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1939 to 1948*, of Queensland, as duly qualified medical practitioners:

Windsor, Morgan Francis, M.B., B.S., 1941 (Univ. Sydney), M.S., 1946 (Univ. Sydney), F.R.C.S. (England), 1948, 18 Whyendah Road, Hamilton, Brisbane.
Zeck, Paul Julian, M.B., B.S., 1943 (Univ. Sydney), Brisbane Mental Hospital, Goodna.
Stelling, Barbara Joan, M.B., Ch.B., 1943 (Univ. Leeds), 20 Echlin Street, West End, Townsville.
Sutton, David Harvey, M.B., B.S., 1942 (Univ. Sydney), c.o. Hospitals Board, Cloncurry.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

McFadden, Peter Grant, M.B., B.S., 1948 (Univ. Sydney), Myoora Street, Pymble.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Grieve, Shirley Euphrosyne, M.B., B.S., 1950 (Univ. Sydney), Goulburn District Hospital, Goulburn.
Quinn, John Vincent, M.B., B.S., 1948 (Univ. Sydney), 12 Hebden Street, Lockhart.
Richards, John Grahame, M.B., B.S., 1950 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

The undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Stoddart, Harrold William Downing, M.B., B.S., 1911 (Univ. Adelaide), 26 Partridge Street, Glenelg.
Wilson, John Grenell, M.B., B.S., 1949 (Univ. Adelaide), 287 Unley Road, Malvern.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association:

Hill, Douglas Henry, M.B., B.S., 1949 (Univ. Adelaide), 26 Dequeteville Terrace, Kent Town, South Australia.
Lovell, Keith Everett, M.B., B.S., 1949 (Univ. Adelaide), Royal Adelaide Hospital, North Terrace, Adelaide.

Diary for the Month.

MAY 23.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 24.—Victorian Branch, B.M.A.: Council Meeting.
MAY 25.—New South Wales Branch, B.M.A.: Branch Meeting.
MAY 26.—Federal Council of the B.M.A. in Australia—Brisbane.
MAY 26.—Queensland Branch, B.M.A.: Council Meeting.
MAY 27-JUNE 3.—Australasian Medical Congress (B.M.A.)—Brisbane.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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